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STUDIES OF CEREBRAL FUNCTION IN LEARNING. No. III.—THE MOTOR AREAS.

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IN their first study of the electro-stimulable cortex of the dog, Fritsch and Hitzig [5] ascribed to it the function of control of voluntary movement, although they recognized that its destruction resulted in a difficulty rather than a complete loss of ability to make voluntary movements. The latter fact was largely disregarded by the investigators following them and the view that the fibres descending from the stimulable area form the chief efferent path from the cortex was adopted by Ferrier, Carville and Duret, Luciani, Bechterew, and other early workers, so that this theory has come to be accepted as one of the most firmly established facts of cerebral localization. From the first, however, certain facts, derived chiefly from studies of recovery from paralysis following lesion to the motor area, have seemed incompatible with this view. Fritsch and Hitzig emphasized the importance of other descending tracts, and Carville and Duret [4] showed that recovery from hemiplegia is not due to the vicarious functioning of the uninjured stimulable area. The belief that recovery from paralysis is only a revival of depressed reflex functions and that the power of voluntary movement is permanently abolished by complete destruction of the motor area was disproved by Rothmann [16] who observed the formation of motor habits in monkeys after destruction of the stimulable area of one hemisphere and the pyramidal and rubrospinal tracts of the opposite side.

Such experiments show that the extra-pyramidal fibres are capable of mediating complex activities of the habitual or voluntary type but

leave undecided the question whether this is their normal function or merely a vicarious function due to the destruction of the stimulable cortex. In a previous study of the rôle of various parts of the cerebrum of the white rat in the formation and retention of visual habits the writer found that the habit of discrimination of light intensities survived the total destruction of the stimulable area. Injury to an area on each occipital lobe corresponding to Brodmann's *area striata* [2] resulted in the complete loss of the visual habit, but any other third of the cortex might be destroyed without in the least disturbing the animal's ability to make the discrimination accurately. The data reported in that study seem to prove that the conditioned-reflex arcs involved in visual habits descend from approximately the same area of the cerebral cortex to which they are projected and do not traverse the cortex to descend from the stimulable areas. The visual habits have cerebral representation, but the supposed motor areas have no direct function in their performance [10].

In higher animals, especially the primates, complete paralysis of voluntary movement may follow destruction of the stimulable cortex, but this does not necessarily imply an interruption of the direct conditioned-reflex arcs. Such an explanation is to be preferred on grounds of simplicity, if contradictory facts do not appear, but disturbances of some of the mechanisms of tonic reinforcement, not in the direct conditioned-reflex path (section of the afferent nerves of a limb, cerebellar injury, &c.), may produce somewhat similar disturbances of voluntary movement and indicate an alternative explanation of the function of the stimulable areas. The demonstration that, in the rat, the stimulable area is not traversed by the conditioned-reflex arcs of visual habits seems sufficient evidence to raise the question of whether the so-called motor area is really a part of the mechanism for voluntary movement or is merely a part of the subsidiary tonic and postural mechanism.

Generalization from the rat to higher forms is complicated, however, by the probable transfer of function from the corpus striatum to the stimulable cortex, with ascent in the evolutionary scale. In the rat, destruction of the stimulable areas produces no detectable motor disturbances, but simultaneous destruction of the motor area and the corpus striatum produces a paralysis which is relatively permanent and which resembles the effect of destruction of the stimulable area in higher forms [10]. This indicates that the function of the stimulable area of primates is represented in the rat by the combined function of

the stimulable area and the corpus striatum.[1] In order that data obtained from the rat may be applicable to higher forms it is necessary, therefore, that both the stimulable cortex and the corpus striatum be considered. My earlier study eliminated only the stimulable cortex from the habit mechanisms. In the experiments reported below I have tested the effects of simultaneous destruction of the stimulable areas and the caudate portions of the corpora striata upon the rat's ability to form and retain visuo-motor habits. The data outlined above make it fairly certain that in the combined lesion we are dealing with structures analogous in function to the stimulable areas of the primates and that conclusions valid for higher forms may be drawn from the experiments.

The chief question which the following experiments were designed to answer is, then, whether or not the structures of the rat's cerebrum, injury to which produces motor disorganization, are directly concerned in the performance of habitual acts. Visuo-motor habits are best suited for such tests, since they are easily acquired and recognized. A visual area in the occipital region is clearly defined and it has been shown that cortical representation of the habit is retained after 1,400 trials of overtraining [11], so that the possibility that the habits are carried out at subcortical levels is ruled out.

The Topography of the Stimulable and Visual Areas of the Cerebrum and of the Corpus Striatum in the Rat.

The stimulable area in the rat includes the antero-dorsal third of the cerebral cortex. It extends from above the anterior margin of the hippocampus forward to the frontal pole, covering the median half of the dorsal convexity in this region. On the frontal pole it extends laterally and then caudally over the inferior orbital surface. Fig. 1 shows a composite map of the area in about twenty-five animals, with the movements elicited by bipolar stimulation. In the majority of animals the area does not extend so far caudally as in the diagram and only movements *a*, *d*, *i*, *j*, and *p* can be elicited.

The visual function seems limited to a small area on the dorsal

[1] Since the pyramidal fibres are scattered throughout the caudate nucleus it is impossible to destroy the latter alone. In earlier work [10] I found one case with extensive degeneration of the right caudate nucleus involving few of the pyramidal fibres and leaving the greater part of the stimulable area intact. This animal showed an unusually rapid and complete recovery from the motor disturbances following operation and led to the tentative conclusion that the striate nucleus and stimulable cortex have interchangeable function.

convexity of the occipital pole. Fig. 2 is a composite diagram of the lesions in nine animals in which habits of visual discrimination persisted after bilateral injury. Detailed descriptions of these animals have been given in previous papers [10, 11]. In each animal the area destroyed included about one-fourth the blackened area of the diagram. Fig. 3 is a composite diagram of the lesions in seven animals which lost the habit of visual discrimination after operation. These two series of animals show clearly the restriction of cortical function in vision to the

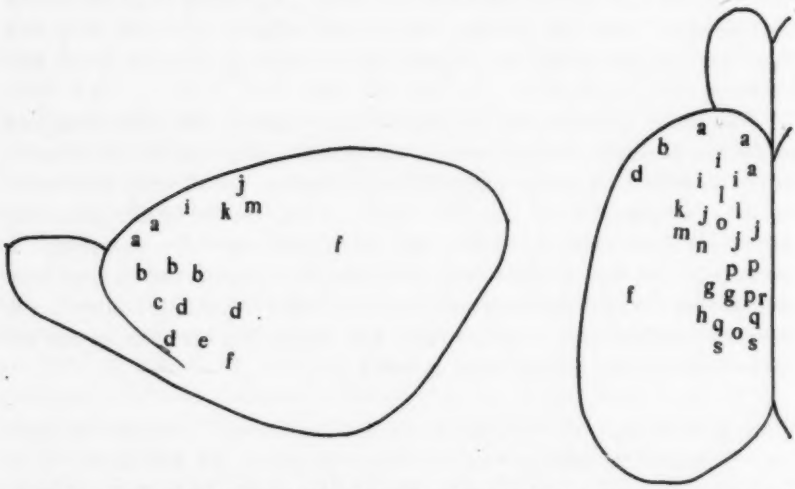


FIG. 1.—Composite diagram of the electro-stimulable points on the cerebral cortex of the rat. *a* = Head turned to opposite side. *b* = Nose retracted. *c* = Vibrissæ moved. *d* = Chewing movements, *e* = Tongue protruded. *f* = Eye closed. *g* = Ear adducted. *h* = Ear erected. *i* = Shoulder drawn forward. *j* = Fore-arm retracted. *k* = Elbow flexed. *l* =Elbow extended. *m* = Wrist flexed. *n* = Fore-arm rotated. *o* = Back flexed to opposite side. *p* = Hind leg drawn forward. *q* = Homolateral leg flexed, ipselateral extended. *r* = Ankle extended. *s* = Tail drawn to opposite side. All movements are contralateral to the hemisphere stimulated, except where indicated.

occipital pole and the survival of the habit after destruction of the motor area. The area included in fig. 3 corresponds roughly to Brodmann's *area striata* in other rodents [2].

The corpus striatum in the rat is very large in proportion to the volume of the cerebral cortex. Fig. 4 shows it in frontal and horizontal sections through the levels used in later diagrams. The caudate nucleus consists of masses of cells scattered among the fibres of the internal capsule and is nowhere distinct from the descending fibres. The lenticular portion is relatively free from pyramidal fibres. The

experimental lesions described below are restricted to the caudate nucleus and this is also the part whose injury produces paralytic symptoms [10, 12].

Experimental Methods.

Training.—For the study of visual habits a discrimination box of the type designed by Yerkes was used. It consists of a central compartment opening into two parallel alleys which lead by lateral passages

Fig. 2.—Composite diagram of the lesions in animals which retained the habit of visual discrimination after operation. The habit survived the destruction of any third of the blackened area.

Fig. 3.—Composite diagram of the lesions in seven animals which lost the habit of visual discrimination after operation. The area which was destroyed in all is shown in solid black.

to compartments in which food is placed. At the end of each alley is a small electric bulb, 5-watt frosted "Mazda," visible from the discrimination compartment. One bulb is lighted and the other darkened in irregular alternation and the animal is trained to choose the illuminated alley, receiving food there and punishment in the darkened one. Training was continued with ten trials per day by the usual method of irregular alternation until the animals made thirty consecutive trials without entering the darkened alley. Controls were intro-

duced to assure that the reaction was to the light and not to accidental cues.

Operative methods.—Under ether anæsthesia the skull was pierced on each side of the median line at the fronto-parietal suture and the bone clipped away to make an opening, about 2 by 6 millimetres, above the caudate nucleus. Through this opening a small electric cautery, heated to redness, was plunged to a measured depth, then drawn slowly back and forth at a nearly constant depth throughout the length of the

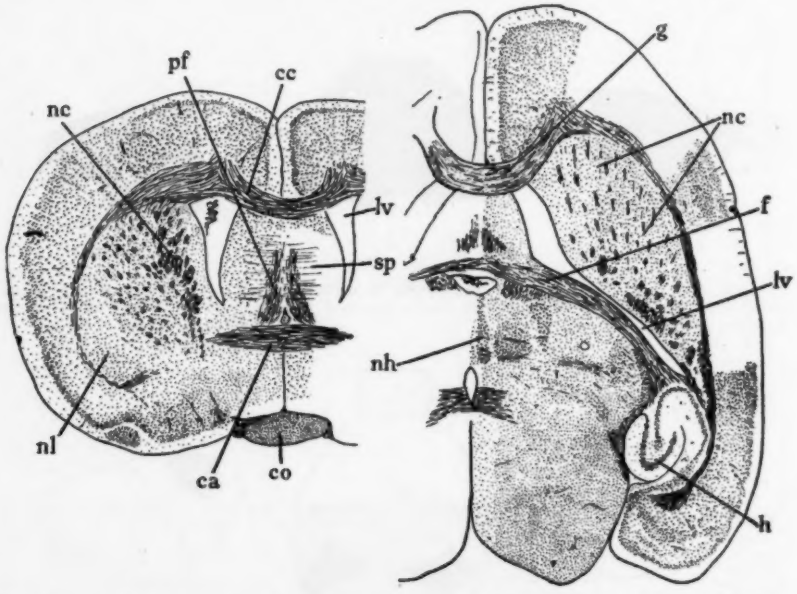


FIG. 4.—Frontal and horizontal sections through the corpus striatum at the levels of its maximum area. *c.a.* = Commissura anterior. *c.c.* = Corpus callosum. *p.f.* = Columna fornicis. *c.o.* = chiasma opticum. *f.* = fornix. *g.* = Genu corporis callosi. *h.* = Lobus hippocampus. *l.v.* = Ventriculus lateralis. *n.c.* = Nucleus caudatus. *n.h.* = Nucleus habenulæ. *n.l.* = Nucleus lentiformis. *sp.* = Septum pellucidum.

caudate nucleus. A fine scalpel was next passed from the posterior margin of the skull opening to the base of the olfactory bulb and drawn back and forth with its point in contact with the floor of the cranial cavity, so as to sever the frontal pole from the remaining cortex. In the majority of cases this method resulted in the complete destruction of the stimulable areas and in extensive lesions to both caudate nuclei. When hæmorrhage was stopped, the scalp was closed with interrupted sutures and covered with a cotton-collodion dressing.

Retention tests.—Animals trained before operation were tested in the problem box as soon as their general condition permitted; usually twenty-four hours after operation. In these tests for retention of the habit the same method was employed as in training, except that no punishment was given for errors during the first fifty trials; that is, the retention test is essentially a retraining in the habit and evidence for retention consists in the reappearance of accurate discrimination in significantly less time than was required for the original learning.

When a habit is lost after operation the question of post-operative shock must be considered. In earlier papers I have discussed methods of distinguishing between loss due to shock and that due to destruction of a functional area. In the present experiments positive evidence for retention following operation was obtained in every case, so that the question of shock does not enter, save as an explanation of the few errors made in the trials immediately following operation.

Reconstruction of lesions.—After completion of the retention tests the brains were removed and serial frontal sections stained with iron hæmatoxylin were prepared. Camera outlines of sections at intervals of one half millimetre were made and the extent of the lesions indicated on them. The positions of the sections were determined from internal structures and indicated on diagrams of these structures projected to the surface of the brain. The dimensions of the lesions were then transferred with proportional dividers to the diagrams, the points so determined connected by lines and the areas inked in. The reconstructions were finally verified by reference to the sections.

Only easily recognizable lesions, absorption or complete degeneration of the cortex and subcortical nuclei were recorded. The diagrams, therefore, represent the minimal destruction produced by the operations. In all cases, probably, larger areas than those shown were destroyed, but, since the experiments deal with the non-function of given areas and do not seek to localize functions accurately, this fact does not invalidate the results.

Graphic and tabular presentation of results.—In the preparation of the figures a uniform system has been followed. Diagrams of the superficial lesions made from camera outlines of an average brain are given at the top of the figure. Below at the left is a diagram of a horizontal section through the cerebrum at the level of the maximum area of the caudate nucleus. Practically all of the ganglion cells of that nucleus are included within the dotted line, the posterior horn being made up almost altogether of the fibres of the internal capsule.

At the right, below, is a camera outline of a frontal section through the cerebrum at the level of the anterior commissure (except in fig. 12 where the section lies at the level of the anterior horns of the lateral ventricles). In these outlines complete replacement of nervous structure by scar tissue or amorphous material is indicated in solid black; marked degeneration with some nerve elements intact is shown by stippling.

In the records of training and retention the "number of trials required for learning" represents the number of times that the animal traversed the problem box from the starting compartment to the food, before thirty consecutive trials were made without entrance into the dark alley. Ten trials were given each day in training and retention tests except where indicated. The records of these trials are given as horizontal rows of figures, each number representing the number of trials, in the ten of that day's practice, in which errors were made. Thus in the record of No. 17, 4 : 2 : 1 : 2, &c., means that on the first day four of ten trials contained errors, on the second two of ten, &c. Five errors in ten trials shows no discrimination. When no error is made for thirty trials, it is rare that errors occur in later trials unless the animal is disturbed by being roughly handled or by some new elements in the situation, such as those introduced by scrubbing the problem box.

The Formation of Habits after Destruction of the Stimulable Areas and the Caudate Nuclei.

The formation of maze, latch-box, and visual habits after destruction of the stimulable area in the rat has been reported in earlier papers [10, 12]. In the present study I have attempted to train animals after destruction of both the stimulable areas and the caudate nuclei. Only three animals have survived the operation long enough to give evidence of learning and they have formed only simple habits. The individual records of these animals follow.

No. 17.[1] Adult male.—The cautery was plunged into each caudate nucleus and the frontal pole of each hemisphere was incised. Training for visual discrimination was begun eight days after operation. For several days he spent most of the time climbing to the top of his cage and falling back to the floor. When placed in the problem box he promptly climbed out and repeated this more than two hundred times in spite of severe punishment. He

[1] For convenience in reference these animals are numbered consecutively with others of the visual series which have been reported earlier [10, 11].

was then placed in the left-hand feeding compartment in contact with the food, and began to eat immediately. By the process of removing him a few inches farther from the food at each trial, he was trained to find his way through the training box from the starting compartment to the food. He was then given regular training in visual discrimination for seven days. In this time he showed no evidence of visual discrimination, but formed a position habit toward the left alley, which was fixed in a normal learning curve, with errors (turns to the right) as follow, in successive tens of trials:—

4 : 2 : 1 : 2 : 1 : 0 : 0.

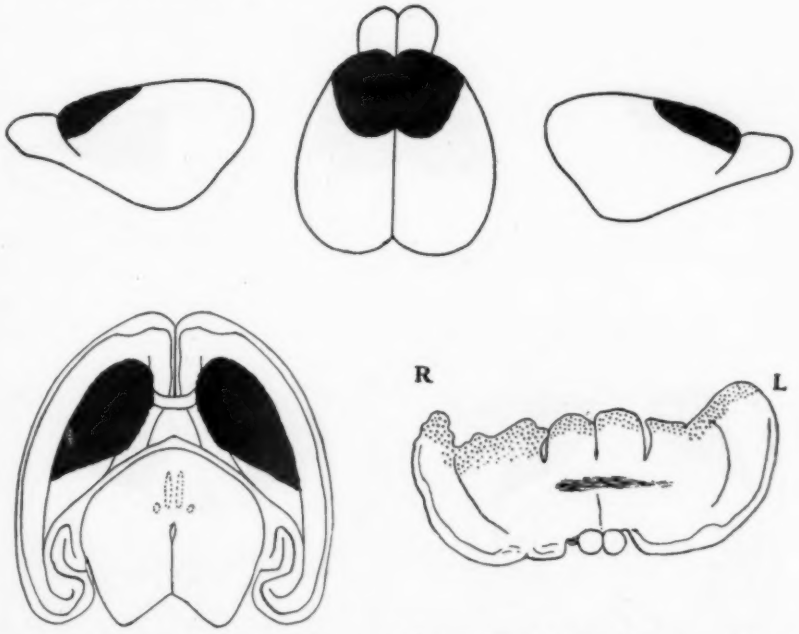


FIG. 5.—Extent of lesions in No. 17. In this and the following diagrams a uniform arrangement has been adopted. Above, the lesions to the cortex are shown in lateral and dorsal aspects. Below, at the left, is a diagram of the horizontal extent of the lesions to the caudate nuclei, in this case embracing the entire extent of both nuclei. At the right is a camera outline of a frontal section at the level of the anterior commissure, showing the depth of the lesion. In these outlines degenerated areas are stippled and scar tissue, which does not appear in this one, is shown in solid black. The outlines of the frontal sections are reversed, the right side appearing on the left. The diagrams are correctly orientated.

Ten days after operation he developed a marked spasticity and died on the eleventh day. Autopsy showed the wound infected, with extensive abscesses in both hemispheres, extending into the lateral ventricles.

Extent of lesions (fig. 5).—Right hemisphere: The entire cortex of the dorsal surface of the hemisphere, from the frontal pole to the level of the

pillars of the fornix, is replaced by a cyst. The dorsal half of the striate nucleus, including all of the caudate nucleus, is absorbed. The lenticular nucleus is uninjured. Left hemisphere: The lesion is identical with that on the right.

No. 18. Small adult male.—The frontal lobes were transected and the caudate nuclei cauterized. For eleven days the animal showed marked disturbances of movement, rotating to the right, so that he was unable to eat without assistance. By the twelfth day this trouble had partially cleared up, so that he could walk in a straight line—although he still tended to rotate

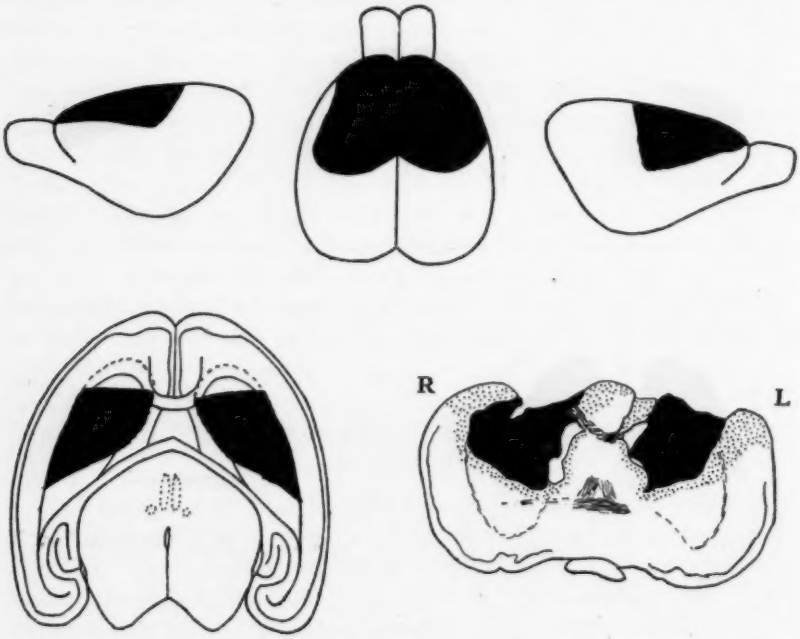


FIG. 6.—Extent of the lesions in No. 18. Arranged as in fig. 5.

when eating. Training in visual discrimination was begun on the fourteenth day after operation. At first he had great difficulty in making the turns in the training box and in finding food, since the operation rendered him anosmic. In five days (fifty trials) he formed the somæsthetic-motor habits of the training box, so that he could go directly from the starting compartment to the food, correcting the error and orientating promptly when he failed to find food in the darkened alley. He still had some difficulty in recognizing food, and would eat shavings and fæces in the neighbourhood of the food dish, but not elsewhere. He died twenty-three days after operation.

Extent of lesions (fig. 6).—Right hemisphere. The lesion to the cortex

extends as a diagonal incision from the level of the thalamico-mamillary tract to the frontal pole, destroying all of the cortex of the dorsal convexity and extending over the superior part of the orbital surface. The caudate nucleus is completely destroyed from the level of the anterior commissure to its posterior margin. The thalamus is invaded at the level of the anterior and lateral nuclei. Left hemisphere: The lesion to the cortex is similar to that on the right, but does not extend so far laterad. The posterior part of the caudate nucleus is destroyed, as on the right. The anterior and lateral thalamic nuclei are destroyed.

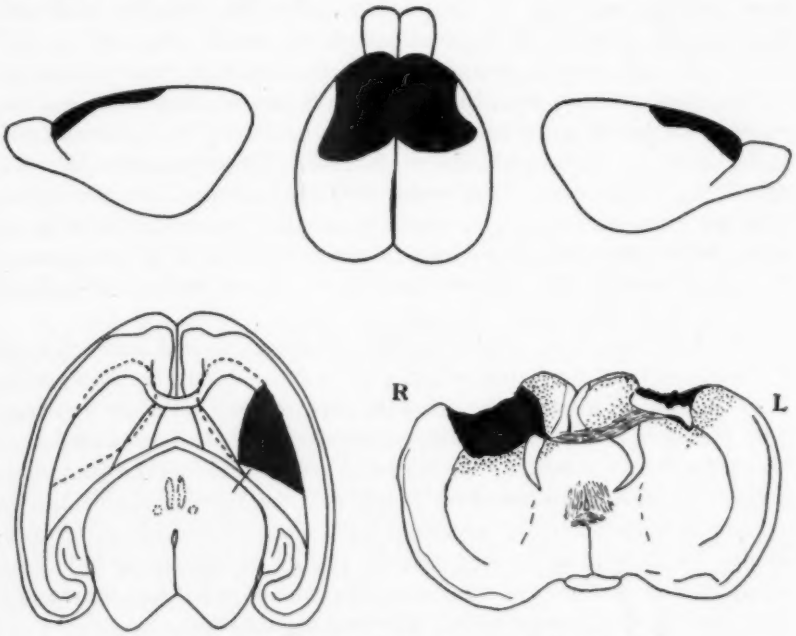


FIG. 7. --Extent of the lesions in No. 19. Arranged as in fig. 5.

No. 19. Large adult male.—The frontal lobes were transected and the caudate nuclei cauterized. Training in brightness discrimination was begun ten days after operation. The habit was formed in 130 trials with errors in successive tens of trials distributed as follows:—

6 : 6 : 2 : 5 : 5 : 2 : 1 : 3 : 1 : 1 : 0 : 1 : 1 : 0 : 0 : 0.

Extent of lesions (fig. 7).—The dorsal convexity of both hemispheres was destroyed from the level of the thalamus to the frontal pole. The orbital surfaces and olfactory tracts were not injured. The lateral portion of the right caudate nucleus was destroyed from the level of the knee of the corpus callosum to its posterior limit. The left nucleus was uninjured.

The data from these animals, in conjunction with that presented in earlier papers, establishes the fact that the rat may form somæsthetic motor habits in the absence of the stimulable cortex and of the caudate nuclei. I have shown that destruction of the stimulable area alone does not lead to an appreciable reduction in the rate of formation of visual habits, and that a difficult latch box, the "double platform box," may be learned at normal rate after complete destruction of the stimulable areas and severe injury to one or both caudate nuclei [10]. Of the three animals reported in the present study, No. 19 alone survived long enough to allow of acquirement of the visual habit and in this animal the left caudate nucleus and part of the right were uninjured. In the other two, however, the stimulable cortex was destroyed and the caudate nuclei were either destroyed, as in No. 17, or severed from their thalamic connections, as in No. 18. These animals survived only a short time, but both gave unmistakable evidence of the formation of simple somæsthetic-motor habits. From the data on the retention of visual habits after the same operation, presented in the following section, it is probable that the animals would have formed the visual habit if training could have been continued.

The problem of the relation of the extent of cerebral destruction to the complexity of the habits which may be formed must be reserved for later discussion when more adequate experimental data are available. The interest of the present study is chiefly in the question of whether or not habitual "voluntary" movements are mediated by the stimulable areas. These experiments show clearly that neither the stimulable area nor the caudate nuclei are necessary for the acquirement of such movements. This is in agreement with the results of Rothmann [16] with monkeys and justifies the conclusion that the so-called motor areas are not essential to the acquirement of voluntary activities.

This at once suggests the more important question of whether under normal conditions the motor areas are directly functional in the performance of habitual reactions. This problem has been attacked by destroying the structures after a complex habit had been acquired and testing for retention of the habit after the operation.

The Retention of Visuo-motor Habits after Destruction of the Stimulable Areas and the Caudate Nuclei.

For tests of retention after operation the animals were trained in the discrimination box until they made thirty consecutive trials without an error. When this record was attained the problem was considered

learned, but in some cases training was continued for from 30 to 60 additional trials, as it was not always convenient to operate earlier. Since I have found that over-training up to 1,400 trials does not influence cortical localization, this additional training is not objectionable. When the problem was learned the animals were subjected to operation and their retention of the habit was tested as soon as their physical condition permitted.

In training ten trials were given daily. When the animal entered the darkened alley he was punished and the trial was recorded as an error, although he was allowed to turn back and pass through the illuminated alley to the food. In the retention tests the same method was used except that the animals were not punished. In the records given below the number of errors made in each successive ten trials of training and of the retention tests is given. Five errors are to be expected from chance if the animal is not discriminating. Fewer than four errors in ten trials suggest discrimination, and no error in thirty consecutive trials usually means that the animal will discriminate accurately thereafter. The individual records of the animals follow.

No. 20. Large male.—Trained in visual discrimination. Sixty trials were required for learning, with errors in successive tens of trials distributed as follows:—

7 : 6 : 2 : 5 : 3 : 2 : 0 : 0 : 0 : 0 : 0 : 0.

The frontal lobes were transected and the caudate nuclei cauterized. The animal was first tested three days after the operation. He was slow and spastic, but made no error in twenty consecutive trials. For the next three days he was in an excited state and showed great fear, either refusing to run or dashing through the training box at random and paying no attention to the food. On the eighth day he was again normal in behaviour and gave clear evidence of discrimination.

Post-operative retention tests; errors in successive tens of trials on consecutive days after operation:—

0 : 0 : (frightened, 6 : 4 : 3) 0 : 1 : 0 : 1 : 0 : 0.

Extent of lesions (fig. 8).—Right hemisphere: the lesion includes all of the cortex of the dorsal convexity from the level of the thalamus to the base of the olfactory bulb. It extends over the orbital surface at the level of the caudate nucleus where the cautery passed through the external capsule. The caudate nucleus is destroyed from the level of the anterior commissure to its posterior limit. There is some injury to the anterior thalamic nuclei. Left hemisphere: the lesion to the cortex is similar to that on the right, but does not extend on the orbital surface. The injury to the caudate and thalamic nuclei is similar to that on the right.

This animal showed unmistakable evidence of retention after

destruction of almost the entire stimulable cortex and of the posterior two-thirds of both caudate nuclei.

No. 21. Small female about 90 days old.—Trained in visual discrimination. Ninety trials were required for learning, with errors in successive tens of trials distributed as follows:—

8 : 5 : 5 : 3 : 2 : 3 : 0 : 4 : 1 : 0 : 0 : 0 : 1 : 1 : 0 : 0 : 0.

The frontal lobes were transected and the caudate nuclei cauterized. For the first week after operation the animal was unable to walk except in 6-inch circles and could not make her way through the discrimination box. By the

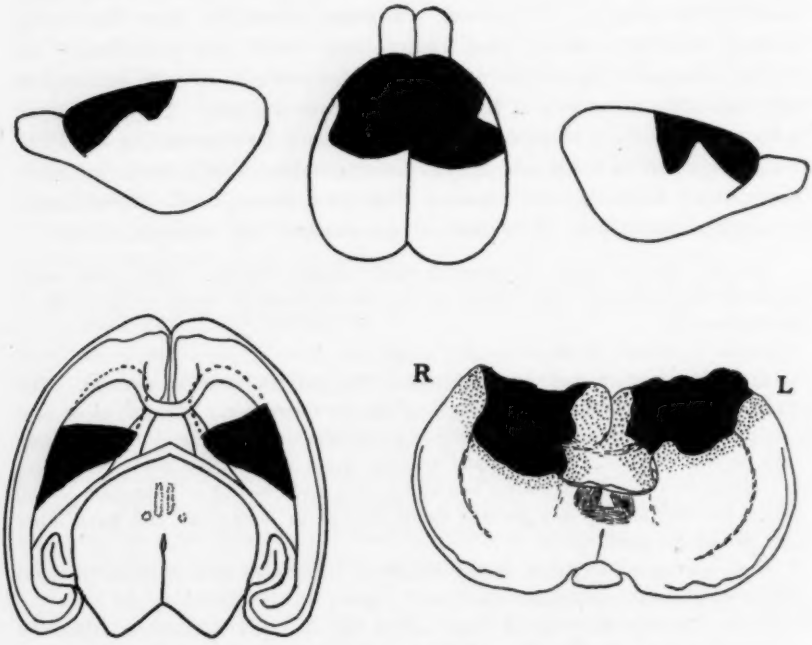


FIG. 8.—Extent of the lesions in No. 20. Arranged as in fig. 5.

ninth day after operation the motor disturbance had cleared up so that she could walk without rotation, although she still rotated when attempting to change direction and could not turn her body to the left. Retention tests were begun at this time and the animal at once gave certain evidence of discrimination. The tests were continued for twelve days. Throughout this time she was unable to turn to the left but compensated by rotating 270 degrees to the right wherever the problem box demanded a turn of 90

degrees to the left. Fig. 14 shows some of her simpler paths in traversing the problem box.

Post-operative retention tests; errors in successive tens of trials on consecutive days.:—

1 : 1 : 4 : 3 : 0 : 1 : 0 : 1 : 1 : 0 : 0.

Extent of lesions (fig. 9.)—All of the cortex of both hemispheres from the level of the thalamus to the base of the olfactory bulb is destroyed. The right caudate nucleus is completely destroyed except for a small antero-medial region laterad to the forceps of the corpus callosum. The left caudate nucleus

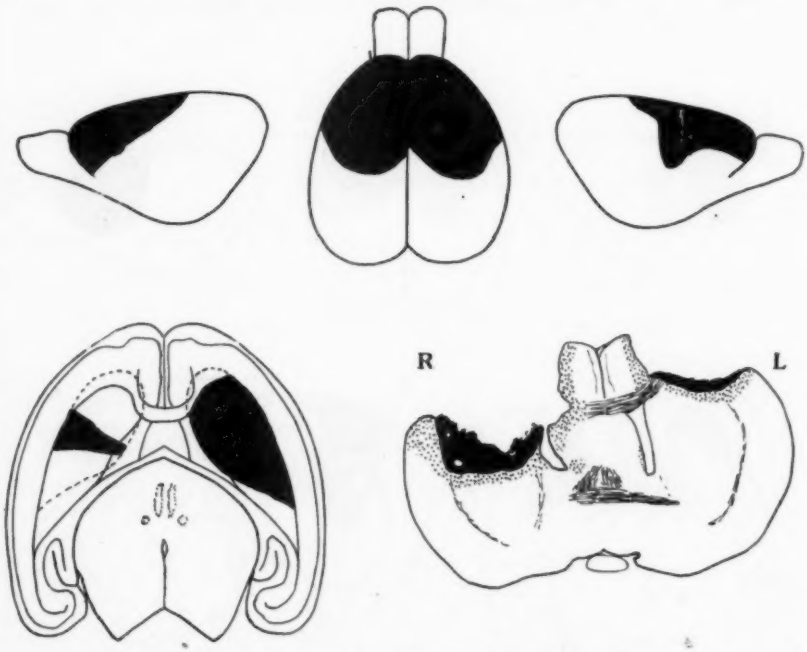


FIG. 9.—Extent of the lesions in No. 21. Arranged as in fig. 5.

has a superficial transverse lesion at the level of the anterior commissure. The right anterior thalamic nucleus is invaded.

After destruction of the stimulable cortex and severe injury to both caudate nuclei, resulting in marked motor disorganization, this animal gave certain evidence of retention of the habit of visual discrimination.

No. 22. Large male.—Trained in visual discrimination. Eighty trials

were required for learning, with errors in successive tens of trials distributed as follows:—

7 : 7 : 4 : 2 : 1 : 1 : 0 : 1 : 0 : 0 : 0 : 0 : 0.

The frontal lobes were transected and the caudate nuclei cauterized. The animal was stuporous for the first day after operation, but on the second day was in fair condition and reacted promptly to the problem box, although tending to rotate to the left. On this day he made four errors in twenty trials, but the errors were obviously due to failure to compensate for the motor difficulty and his behaviour in the discrimination compartment clearly indicated discrimination. On the following and later days he made no errors.

Post-operative retention tests; errors in successive tens of trials:—

2 : 2 : 0 : 0 : 0 : 0.

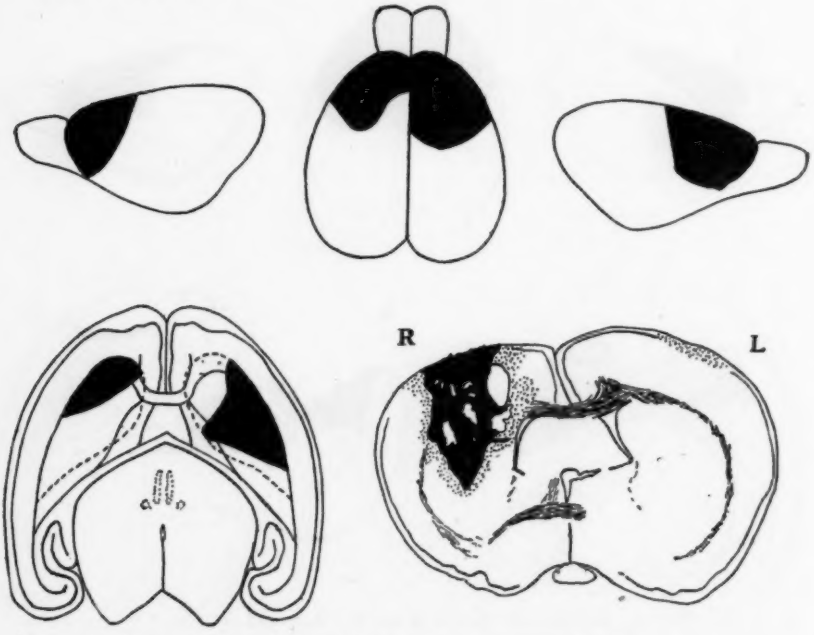


FIG. 10.—Extent of the lesions in No. 22. Arranged as in fig. 5.

Extent of lesions (fig. 10).—Right hemisphere. The lesion extends from the level of the thalamus to the base of the olfactory bulb, separating all of the cortex above this plane from the underlying structures. The cortex of the orbital surface was destroyed by the cautery. The lateral portion of the caudate nucleus is destroyed by a lesion which embraces the full width of the nucleus at the level of the anterior commissure and grows narrower cephalad and caudad to this level. Left hemisphere: The incision extends

from the level of the anterior commissure to the base of the olfactory bulb, destroying all of the cortex above this plane. All of the caudate nucleus in front of the knee of the corpus callosum is degenerated; the posterior part is uninjured.

This rat retained the visuo-motor habit after extensive but incomplete lesions to the stimulable cortex and caudate nuclei.

No. 23. Small adult male.—Trained in visual discrimination. Forty trials were required for learning, with errors in successive tens of trials distributed as follows:—

7 : 5 : 2 : 4 : 0 : 0 : 0 . 0 : 0 : 0 : 0 : 2 : 0 : 0 : 0.

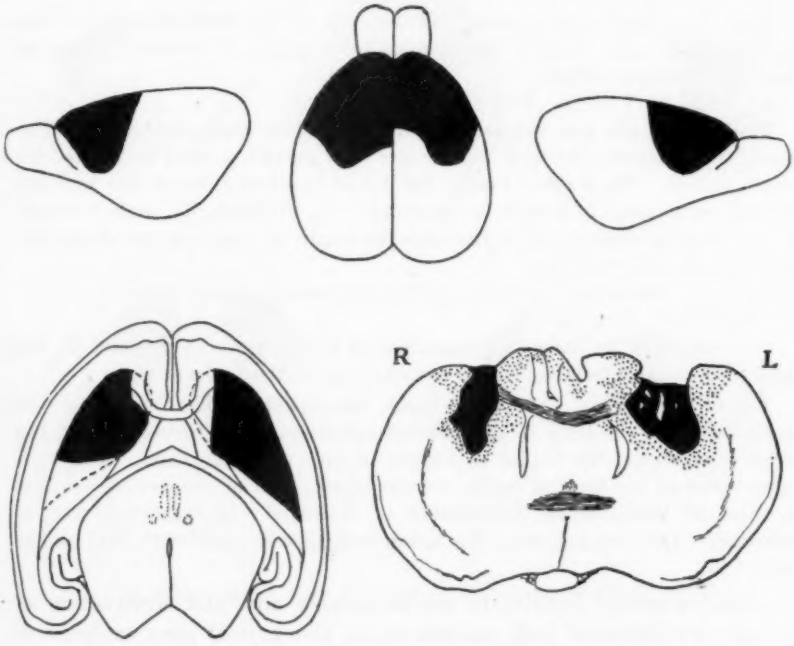


FIG. 11.—Extent of the lesions in No. 23. Arranged as in fig. 5.

The frontal lobes were transected and the caudate nuclei cauterized. For three days after operation the animal's eyes were closed and he could not be tested. On the fourth day he was in good condition and gave clear evidence of discrimination.

Post-operative retention tests; errors in successive tens of trials:—

2 : 1 : 0 : 0 : 0.

Extent of lesions (fig. 11).—Right hemisphere: The lesion to the

cortex begins at the level of the thalamus as a narrow band and broadens cephalad to include all of the cortex of the frontal pole. The lesion to the corpus striatum extends from the caudal limit of the nucleus throughout its length. All of the lateral half of the nucleus is destroyed, but the lesion probably did not involve the median part in front of the anterior commissure. The depth of the lesion is the same throughout its length, involving a little more than the dorsal third of the corpus striatum. Left hemisphere: The lesion to the cortex is practically identical with that on the right. The greater part of the caudate nucleus is destroyed, only the extreme poles remaining uninjured.

This animal showed perfect retention to the visuo-motor habit after nearly complete destruction of the motor areas.

No. 24. Small adult male.—Trained in visual discrimination. One hundred trials were required for learning with errors in successive tens of trials distributed as follows:—

4 : 5 : 3 : 5 : 1 : 3 : 4 : 0 : 2 : 2 : 0 : 0 : 0 : 0.

The frontal pole was transected and the caudate nuclei cauterized. The animal was stuporous for forty-eight hours after operation, then was tested for discrimination. He reacted slowly, but found his way through the problem box and gave some indication of discrimination, although he made frequent errors. On the fourth day of the tests he began to discriminate accurately and made no further errors.

Post-operative retention tests; errors in successive tens of trials:—

3 : 5 : 3 : 0 : 0 : 0 : 0.

The fact that the habit was reacquired in thirty trials, in contrast to the hundred of original training, shows retention rather than relearning.

Extent of lesions (fig. 12).—Right hemisphere. The lesion to the cortex begins at the level of the anterior commissure and includes all of the dorsal convexity of the frontal pole except a narrow median band. The hind leg and part of the fore leg region remain intact. All of the caudate nucleus in front of the anterior commissure is destroyed; its posterior part is uninjured. Left hemisphere: The lesion is almost identical with that on the right.

After extensive injuries to the stimulable area and destruction of the anterior halves of both caudate nuclei this animal gave evidence of retention.

No. 25. Small adult female.—Trained in visual discrimination. One hundred and ten trials were required for learning, with errors in successive tens of trials distributed as follows:—

4 · 5 · 4 : 5 : 2 : 3 : 5 : 3 : 1 : 1 : 1 : 0 : 0 : 0 : 0 : 0 : 0.

The frontal pole was transected and the caudate nuclei cauterized. For three days after operation the animal was unable to walk except in a very narrow circle to the right and could not get through the problem box. On the

fourth day she still rotated but was able to find her way through the box by keeping near the partitions, and made only one error in ten trials. On the following day she developed pronounced fear reactions and climbed out of the training box more than one hundred times. She was finally induced to make ten trials, but made six errors. Training was discontinued for five days. When she was next tested the fear had disappeared and she showed perfect discrimination.

Post-operative retention tests; errors in successive tens of trials :—
1 : 6 : 0 : 1 : 0 : 0 : 0.

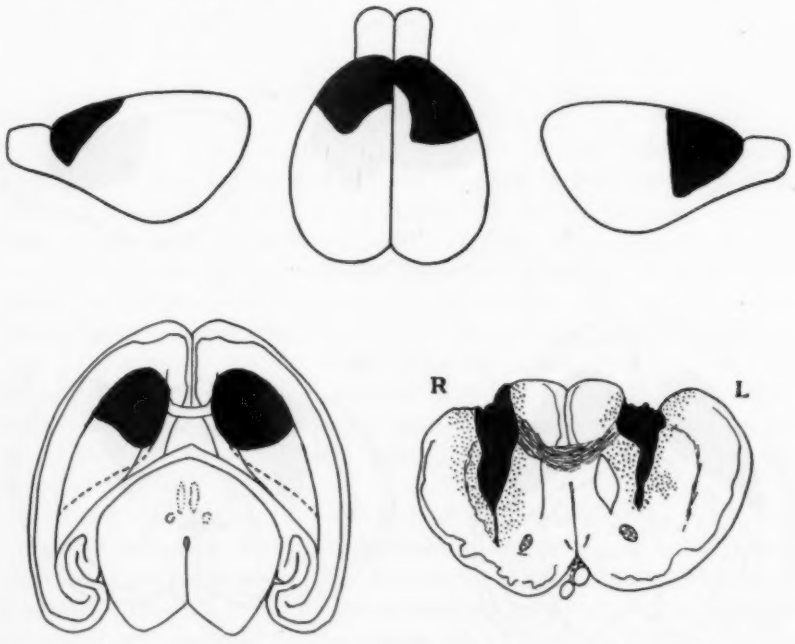


FIG. 12.—Extent of the lesions in No. 24. Arranged as in fig. 5.

Extent of lesions (fig. 13).—Right hemisphere: The lesion to the cortex begins at the level of the commissura habenulæ and extends to the base of the olfactory bulb, including all of the motor cortex except a part of the hind leg area. The cautery passed throughout the length of the caudate nucleus and the entire nucleus is degenerated. Left hemisphere: The lesion to the cortex is similar to that on the right, but misses the antero-lateral surface of the frontal pole, and does not extend so far over the orbital surface. The caudate nucleus is entirely degenerated.

Practically the entire stimulable cortex and both caudate nuclei

were destroyed in this animal. There was clear evidence of retention following the operation.

This group of animals is not selected, but comprises all that survived the operation long enough to allow of retention tests. Every one of them gave evidence of retention early in the tests. Table I gives the number of trials and the number of errors, in learning and and in the post-operative retention tests. The average number of trials required for learning is eighty. Only 33·3 trials on the average

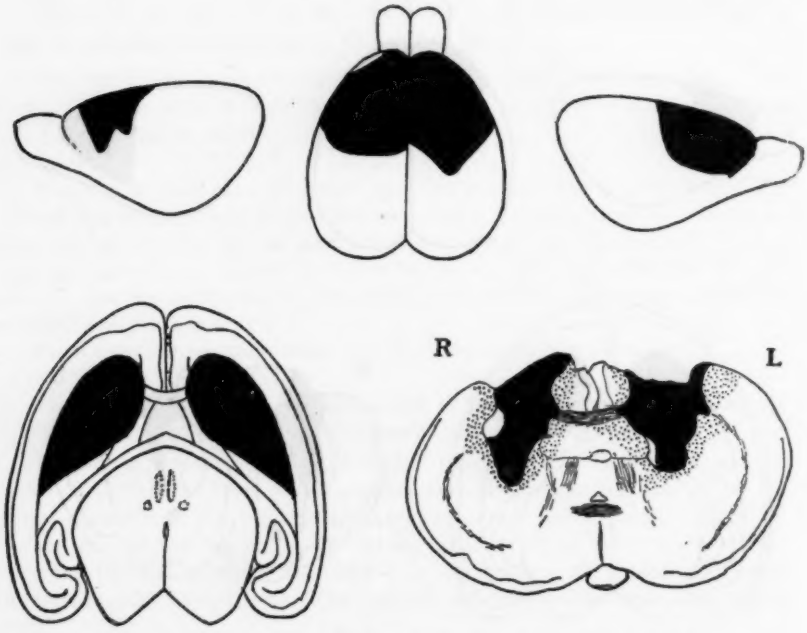


FIG. 13.—Extent of the lesions in No. 25. Arranged as in fig. 5.

were required after operation to reach the same degree of accuracy in discrimination. Errors were made in an average of 26·6 trials in learning and in only 6·3 trials in the retention tests. These figures include errors obviously due to motor disturbance and fright following operation. Thus, No. 21 was never able to turn directly to the left and all her errors seemed due to failure to compensate for the motor difficulty; No. 24 was slow and stuporous during the time when errors were made; six of the eight errors made by No. 25 were made

on a day when the animal was too disturbed to eat. If allowance is made for such errors, retention in every case was practically perfect.

TABLE I.—Numbers of Trials and of Errors preceding the First Thirty Consecutive Trials without Error in Learning and Post-operative Retention Tests for Animals operated after Training.

	Training		Retention tests	
	Trials	Errors	Trials	Errors
No. 20	60	25	0	0
No. 21	90	31	90	12
No. 22	80	23	20	4
No. 23	40	18	20	3
No. 24	100	29	30	11
No. 25	110	34	40	8
Average	80	26·6	33·3	6·3

The small operative field presented by the rat's brain and the necessity for avoiding hæmorrhage on the floor of the cranial cavity or injury to the optic nuclei of the thalamus make the complete destruction of the caudate nuclei difficult. It was accomplished only in No. 25. In this animal also the entire motor area, except the posterior part of the hind leg region, was destroyed. The animal showed motor and emotional disturbance, but gave certain evidence of retention of the habit. In No. 20 the posterior halves of both nuclei were destroyed; in No. 24, the anterior half; in the others the lesions were irregular, but even more extensive. The lesions to the cortex in all cases included the neck, fore-leg, and the greater part of the hind-leg regions.

Earlier experiments [10] had shown that no part of the stimulable cortex is necessary for the performance of the visual habit. The alternative explanations of this fact were: (1) that the striate nuclei function either vicariously for the stimulable cortex when the latter is destroyed, or normally as the source of efferent impulses from the cerebrum; or (2) that the supposedly motor regions of the cerebrum do not lie in the direct path of conditioned-reflex arcs and are of only secondary importance in the performance of voluntary movements.

The foregoing results seem to prove that the second hypothesis is correct. In the absence of the stimulable cortex, injury to the caudate nuclei does not affect retention of the visual habit and perfect discriminations may appear, even after complete destruction of both nuclei. It seems clear, therefore, that neither the stimulable cortex nor the caudate nuclei are directly functional in the performance of the visuomotor habit.

The experiments have not dealt with the lenticular nucleus and the possibility that this forms a link in the conditioned-reflex arc remains. The data previously presented on the effects of injuries to the striate nuclei [10] indicate that the caudate nucleus and the stimulable cortex together in the rat are equivalent in function to the stimulable cortex of higher forms, since their combined destruction results in relatively irrecoverable paretic symptoms. On embryological grounds there is less evidence for assuming homologous function between the lenticular nucleus and the stimulable cortex than between the latter and the caudate nucleus. In some of the earlier experiments of this series, Nos. 7 and 8 [10], the cerebral cortex and underlying association fibres were almost completely transected at the level of the anterior thalamic nuclei; in the present experiments the greater part of the internal capsule has been destroyed without abolishing visual discrimination. The cortical relations of the lenticular nucleus are not well established, but any occipito-lenticular fibres which may exist were almost certainly destroyed by these operations. It seems very improbable, therefore, that the lenticular nucleus has any more important place in the conditioned-reflex arc than has the caudate nucleus.

The evidence presented in this series of papers all points to the conclusion that in the rat the area striata on the dorsal convexity of the occipital pole is the only cerebral structure which functions in the habit of discrimination of light intensities. The habit may be formed or retained after the destruction of any other part of the cerebrum (with the possible exception of the ectorhinal and inferior temporal regions, which have not been explored but are almost certainly not functional in this habit). This means that the conditioned-reflex arcs which mediate the habit must pass to and from the cortex by way of the occipito-thalamic fibres, that the reintegrations occur within the limits of the visual area, and that long transcortical association fibres do not function in the formation or retention of the habit.

Are the conditions similar for other types of habit? There are no significant data for more complex habits. The poor vision of the rat makes the formation of complex visual habits so slow that I have not been able to carry out similar experiments with them. I have not yet located an auditory area. Certain types of latch-box habits are abolished by injury to the stimulable area [12]. The effective lesions here do not correspond in extent to the stimulable area, but only to the excitable region for the neck and fore limbs, which are used no more than the trunk and hind limbs in the manipulation of the latch. It is

probable that the loss in this case is due to the fact that the frontal region is an important somæsthetic projection area as well as electro-stimulable. The one case for which there is clear evidence is the group of somæsthetic habits of the discrimination box which determine general orientation, finding of food, responses to doors, prompt correction of errors, &c. These survive the destruction of any given third of the cortex [10] and of the subcortical cerebral nuclei. It is possible that they are formed at levels below the cerebrum; it is certain that they are not dependent upon the so-called motor structures.

There is some evidence, to be reported later, that the habit of the "double platform box" [10] is disturbed but not completely abolished by destruction of either the frontal or the occipital regions. This suggests that more complex habits involving diverse sense organs may demand co-ordination of distant portions of the cerebrum and that the visuo-motor habit dealt with in this study is too simple to give a typical picture of cerebral function. It does not, however, indicate any greater importance of the pyramidal tracts in complex habits, and we may conclude that the existing evidence for the rat all opposes the direct participation of the "motor areas" in the performance of any habit.

The Function of the Stimulable Area and the Caudate Nucleus in the Rat.

The evidence that the stimulable cortex in the rat has no direct function in the performance of habitual motor adjustments seems conclusive. What then is the function of this portion of the cortex? Data for a final answer to the question are not available, but some suggestions are given by the motor disturbances following unilateral lesion to the stimulable area and the caudate nucleus.

Immediately following combined unilateral destruction of the caudate nucleus and the stimulable cortex the animal shows a very pronounced rotation toward the injured side. Left undisturbed, he assumes a normal position, but immediately upon stimulation he bends sharply toward the side of the injury and walks in a circle, sometimes so narrow that the head is brought across the hind quarters or trips up the hind feet. The legs of the side opposite the injury are hyper-extended and spastic (fig. 15*c*). This condition may persist for several weeks, but usually clears up within a few days to such an extent that, when the animal is placed in a large open space and allowed to become orientated, he is able to walk in a straight line. But the tendency to

rotate still appears in many situations. When the animal is placed on the floor he turns around several times before taking a direct course. The necessity for avoiding an obstacle frequently results in several complete rotations. The tendency to rotate becomes more pronounced in narrow quarters and in the problem box the animals have a good deal

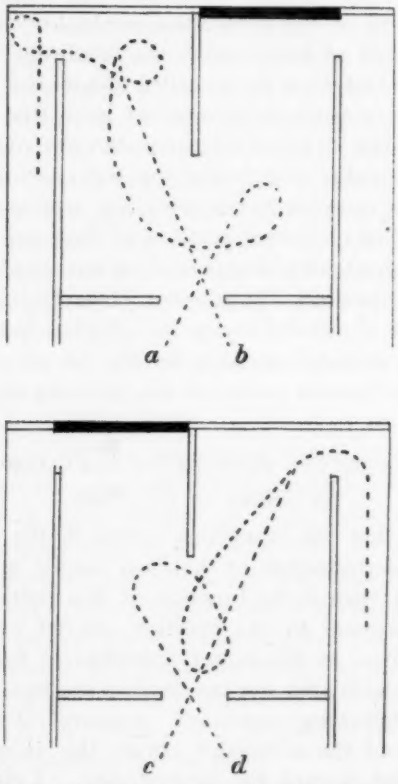


FIG. 14.—Tracings of paths followed by No. 21 in the discrimination box. The animal was unable to turn to the left but compensated for this motor difficulty by rotation to the right, as shown. The discrimination was in each case correct. The path varies with the alley illuminated and with the initial orientation.

of difficulty in making the necessary turns. Fig. 14 shows tracings of the path of animal No. 21 at a time when she was making no errors of discrimination. She was able to walk in a fairly straight line, but any need for change of direction resulted in rotation toward the right. (Only the right caudate nucleus was injured in this animal.) Her path

was usually more complicated than those shown, the turns involving not one but several complete rotations.

In feeding and scratching the animals show similar difficulty in

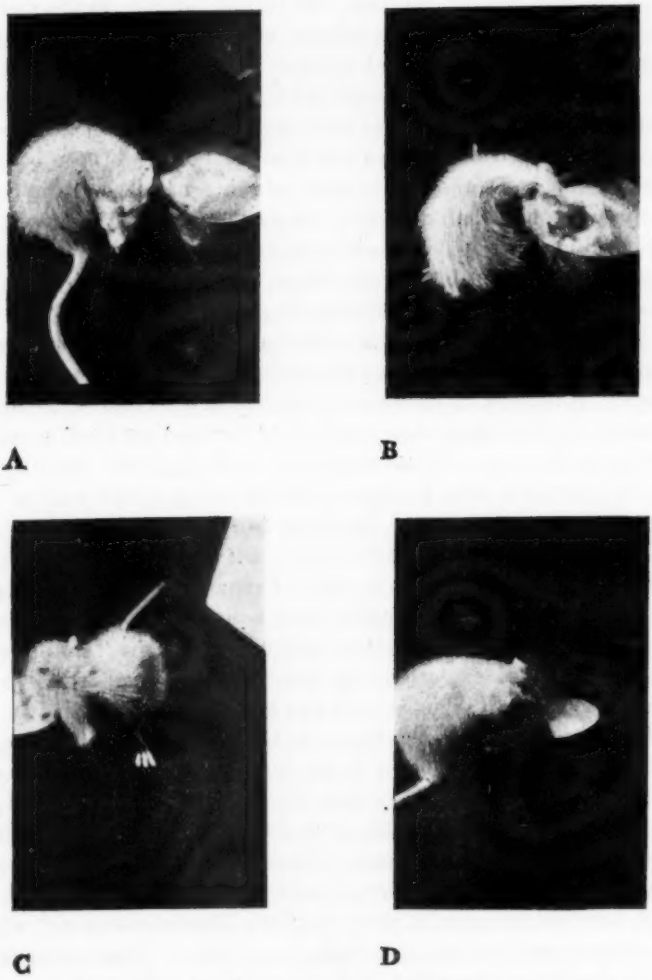


FIG. 15.—The behaviour in feeding of an animal with total destruction of the right stimulable area and caudate nucleus. For explanation see text.

adjustment. Fig. 15 shows various positions assumed by an animal with complete right unilateral destruction of the stimulable cortex and

caudate nucleus, three weeks after operation. She approached the dish, walking in a rather wide circle, but as soon as her mouth came in contact with the food, which stimulates the normal rat to squat and assume a definite feeding position, she turned sharply to the right, the position shown at A. She then rotated several times to the right until by chance her nose was braced against the side of the dish (B). A similar position with hyperextension of the left legs is shown at C. Once such a feeding position has been attained it may be held for some time and a gradual relaxation of the strained posture of the body then appears. Thus, at D, the animal was held in contact with the food for about thirty seconds, then gradually released. She then maintained the normal feeding position for nearly a minute, turning only gradually to the right, as successive bites were taken from the edge of the cube of bread, and finally becoming overbalanced and falling.

In all the activities of such animals, the greatest disturbance of co-ordination appears when some new adjustment to the environment is necessary. Initiation of locomotion, change in direction of locomotion, assumption of a sitting posture, stretching forward for food, scratching, &c., are attended by sudden rotations as though the difficulty were chiefly in gaining a new posture or in initiating a new activity. Yet the activities are initiated and the disturbances are compensated for in ways that indicate that the difficulty is not at the level of the most complex integrations. Thus, in 1917, Franz and the writer reported the case of an animal with complete destruction of the motor cortex and degeneration of one striate nucleus, which learned the "simple maze." The maze used had essentially the same ground plan as the problem box shown in fig. 14. The animal was trained to go constantly to the left. He rotated and fell constantly to the right and at first could not make the turn to the left. He finally learned to grasp the end of the partition with his left hind foot and, with this as a pivot, to swing his body to the left and so enter the food compartment. Fig. 14 shows a more common type of adjustment where the drive toward the goal is maintained in spite of the motor difficulty.

Such adaptations seem to prove that the interference is not with the more complex integrations of voluntary reactions. The compensations rather suggest those described by Luciani [13] for dogs after cerebellar injury, where the tendency to rotate is compensated for by walking with the body curved, and where the lesion admittedly interferes only with reflex tonic control.

The recent discussions of postural reflexes by Sherrington [17] and

Langelaan [9] and of their relation to decerebrate rigidity [Wilson, 19], suggest a possible explanation for the results of destruction of the cerebral motor structures in the rat. The initiation of almost any new activity demands a modification of postural tone, the assumption of a different pattern of tonic contractions, which may then persist almost unaltered for some time during the performance of finer manipulative movements. The behaviour of the operated rats suggests strongly that their chief difficulty is in taking a new posture. Once an activity has been initiated and maintained for a moment even artificially, as when the animal is held in contact with the food, it may persist for a relatively long time until some stimulus to a new posture occurs. The disturbances seem most pronounced in the attempts to perform those activities which require the most complex and unusual postural co-ordinations: scratching and feeding. The compensations for the motor disturbances also suggest that the latter involve less complex, more nearly reflex, integrations than habitual acts. The primary compensations are not by the direct restitution of motor control (recovery from the paresis) but by the acquisition of new patterns of co-ordination which tend to counteract the motor difficulty, much as a normal animal would adapt to a heavy weight attached to one leg. After the destruction of the stimulable cortex the complex adaptive mechanisms are still intact.

These facts argue strongly for a relative independence of the cerebral mechanisms for control of postural activity from those for voluntary movements. The hypothesis which seems best to fit the facts for the rat is that the stimulable area and caudate nucleus are concerned chiefly with the regulation of postural reflexes and that upon these are superimposed the more complex integrations constituting voluntary acts, transmitted to the final common paths by extrapyramidal fibres. This would make the cerebral motor structures an additional stage in the hierarchy of spinal, vestibular, and cerebellar mechanisms for the control of postural reflexes. Such a function has already been ascribed to the corpus striatum by Wilson [18], on the basis of work with monkeys, and the present experiments add to his conclusions only the fact that the stimulable area of the rat is to be classed in function with the striate nucleus rather than with the cerebral habit-mechanisms.

The Function of the Stimulable Cortex in Higher Mammals.

Is such a view of the function of the electro-stimulable cortex contradicted by evidence from the study of higher animals? It is certainly

contrary to the accepted view of its function. In current text-books of physiology and psychology it is frequently stated that the motor area is the (sole) efferent path of voluntary movements from the cortex. Even investigators like Rothmann, who have studied recovery from hemiplegia in animals, look upon the stimulable area as the chief source of the efferent fibres concerned with habitual activity. In speaking of conduction through extrapyramidal paths Rothmann says, "Diese extrapyramidale Leitung dürfte für die Erlernung neuer Bewegungen von grösster Bedeutung sein, während die in festen Besitz des Individuums, übergegangenen gut eingelernten Bewegungen vorwiegend die direkte Verbindung von Grosshirnrinde und Rückenmark, also die corticospinale Bahn, benutzen werden" [16]. Von Bechterew advances a similar view in describing the course of conditioned-reflex arcs across the cortex from the sensory projection areas to the motor area [1].

If this conception is correct for higher forms, it means that a pronounced change in the function of the stimulable cortex has occurred somewhere above the rodents in the evolutionary scale. But such changes in function are rare and in this case the evidence does not seem to show more than that the stimulable cortex of higher forms has taken over a part of the function which the caudate nucleus exercises in rodents.

King [8], using the Marchi method, concluded that the pyramidal tract in the rat is small and unimportant, but Ranson [15] has shown that it is quite large, though made up chiefly of thinly medullated fibres. The area of the tract shown on Ranson's figures is proportionally greater than in primates, so that it cannot be argued that there is any great increase in the anatomical importance of the tract with ascent in the evolutionary scale.

The physiological evidence does not seem sufficient to prove that the pyamidal tracts, even of primates, are the efferent path for impulses to voluntary movement. Fritsch and Hitzig [5] pointed out that the paralysis following destruction of the stimulable area is only partial. Writing of the motor centre they stated that " . . . es ist sicher, dass eine Verletzung dieses Centrum die willkürliche Bewegung des von ihm sicher in einem gewissen Abhängigkeit stehenden Gliedes nur alterirt, nicht aufhebt, dass, also irgend einem motorischen Impulse noch andere Stätten und Bahnen offen stehen um geboren zu werden und um zu den Muskeln jenes Beines zu eilen . . ." Nothnagel observed recovery from the effects of unilateral injury to the motor area and Carville and Duret [4] first showed that the recovery is not due to

the vicarious functioning of the motor cortex of the opposite side. Rothmann [16] showed that in monkeys the extra-pyramidal paths are able to mediate learned activities. Thus it is certain that whatever the normal function of the stimulable cortex in higher mammals it is not absolutely necessary for the performance of voluntary acts.

The chief evidence for the voluntary function of the motor areas comes from the cerebral paralyses in monkeys and man. In them finer manipulative movements are greatly affected and the paralysis is much more severe than in lower mammals, yet even so it is rarely complete. The condition has been characterized as an enormous difficulty rather than an inability to move the paralysed limbs, and there is some evidence that the degree of paralysis varies with the general tonic condition of the organism.

Thus after cauterization of the stimulable cortex on one side the rhesus monkey may show a well-marked hemiplegia. But if he is badly frightened and chased about the room, he uses the limbs of both sides in an apparently normal manner. Left undisturbed, he again lapses into the paralytic condition. Gierlich [6] summarizes other examples of this sort. Similar incidents are occasionally reported for man. A girl with hemiplegia of fourteen years' standing, following diphtheria, with paralysis of the right limbs and marked contractures, tells me that when her brother pushed her from the wharf into a lake recently she used her paralysed limbs actively in climbing out. This patient is mentally retarded and perhaps not reliable. I know of no well-authenticated case of the sort, but evidence of less pronounced effects of emotional disturbance upon paralyses is frequently encountered. Depressing stimuli, such as discussion of the above patient's very distressing home conditions, increase the extent of contracture and limit the power of voluntary movement. Exciting stimuli, on the other hand, will sometimes increase the extent of voluntary movement, and have been used to advantage in re-education (personal communication from Dr. S. I. Franz).

All these facts point to the conclusion that, even in primates, it is not so much the mechanism of voluntary movement which is affected in cerebral paralysis, as some facilitating mechanism whose action is usually essential to the movement, yet is not a part of the direct conditioned-reflex arc. Graham Brown's study of facilitation after section of the connections between pre- and post-rolandic areas [3] shows that the descending fibres from both areas act upon the same final common paths, and that transcortical connections are not necessary for the mutual

facilitation of the areas. Voluntary movements are possible in the absence of the motor areas; there is not conclusive evidence that they function directly in habit; the phenomena of cerebral paralysis can be explained on the assumption that the motor area is postural and facilitating in function; there is clear evidence that the analogous structures in lower forms are not directly concerned with the performance of learned activities. Although not conclusive, these considerations seem sufficient to cast doubt upon the accepted explanation of the function of the motor cortex, and to demand further experimental investigation before we can conclude that the pyramidal tracts form the efferent path for impulses to voluntary movements.

Summary.

It has been shown that the albino rat is able to acquire somæsthetic-motor habits after destruction of the electro-stimulable cortex and the caudate nucleus. Visuo-motor and simple somæsthetic-motor habits which are acquired before the operation are retained after the destruction of these structures and probably after the section of occipito-lenticular fibres. It is clear, therefore, that neither the cerebral-motor areas nor the subcortical nuclei are directly concerned with the performance of learned activities.

Combined destruction of the motor area and the caudate nucleus results in relatively permanent motor disturbances resembling hemiplegia in monkeys. Evidence is given that the difficulty is primarily in assuming new attitudes, and it is suggested that the primary function of the cerebral motor structures in the rat is the regulation of postural reflexes. Existing evidence does not seem to exclude such an explanation of the function of the stimulable cortex, even in man.

References.

[1] Bechterew, W. v. "Die Funktionen der Nervencentra," Jena, 1908.

[2] Brodmann, K. "Vergleichende Lokalisationslehre der Grosshirnrinde," Leipzig, 1909.

[3] Brown, T. G. "The Motor Activation of Parts of the Cerebral Cortex other than those included in the so-called 'Motor' Areas in Monkeys, with a Note on the Theory of Cortical Localization of Function," *Quart. Journ. Exp. Physiol.*, 1916, **10**, 103-144.

[4] Carville, C., et Duret, H. "Sur les fonctions des hémisphères cérébraux," *Archives de Physiol.*, 1875, **7**, 352-490.

[5] Fritsch, G., and Hitzig, E. "Ueber die elektrische Erregbarkeit des Grosshirns, *Arch. f. Anat. u. Physiol.*, Jahrg. 1870, 300-332.

[6] Gierlich, N. "Ueber Symptomatologie, Wesen und Therapie der hemiplegischen Lähmung," Wiesbaden, 1913.

[7] Goltz, F. "Ueber die Verrichtungen des Grosshirns," *Arch. f. d. ges. Physiol.*, 1881, **26**, 1-49.

[8] KING, J. L. "The Cortico-spinal Tract of the Rat," *Anat. Record*, 1910, **4**, 245-252.
[9] LANGELAAN, J. W. "On Muscle Tonus," *Brain*, 1915, **38**, 235-380.
[10] LASHLEY, K. S. "Studies of Cerebral Function in Learning," *Psychobiology*, 1920, **2**, 55-135.
[11] LASHLEY, K. S. Studies, &c., II: "The Effects of Long-continued Practice upon Cerebral Localization," *Journ. Compt. Psychol.*, 1921.
[12] LASHLEY, K. S., and FRANZ, S. I. "The Effects of Cerebral Destruction upon Habit Formation and Retention in the Albino Rat," *Psychobiology*, 1917, **1**, 71-140.
[13] LUCIANI, L. "Human Physiology," London, 1915, vol. 3.
[14] MONAKOW, C. v. "Die Lokalisation im Grosshirn," Wiesbaden, 1914.
[15] RANSON, S. W. "The Fasciculus Cerebro-spinalis in the Albino Rat," *Amer. Journ. of Anat.*, 1912-13, **14**, 411-424.
[16] ROTHMANN, M. "Ueber die physiologische Wertung der cortico-spinalen (Pyramiden-) Bahn," *Arch. f. Anat. u. Physiol.* (Physiol. Abth.), 1907, 217-275.
[17] SHERRINGTON, C. S. "Postural Activity of Muscle and Nerve," *Brain*, 1915, **38**, 191-234.
[18] WILSON, S. A. K. "An Experimental Research into the Anatomy and Physiology of the Corpus Striatum," *Brain*, 1913-14, **36**, 427-492.
[19] WILSON, S. A. K. "On Decerebrate Rigidity in Man and the Occurrence of Tonic Fits," *Brain*, 1920, **43**, 220-268.

A PSYCHOLOGICAL INQUIRY INTO THE NATURE OF THE CONDITION KNOWN AS CONGENITAL WORD-BLINDNESS.

BY LUCY G. FILDES.

THE aim of the investigation about to be described was to discover something of the psychological characteristics of the condition commonly called by the misleading term of congenital word-blindness—a condition which shows itself most clearly in the subjects' extreme difficulty, or even total failure, in learning to read, and appears to be closely related to the various forms of acquired alexia met with commonly as the result of brain injury in later life.

Three explanatory theories, it will be remembered, have been put forward as indicating the nature and cause of this condition, viz.:—

(1) A theory which assumes the existence of definitely localized and circumscribed visual and auditory word-centres in the brain, the destruction or isolation of which will destroy language in either its visual or its auditory aspect;

(2) A theory which interprets word-blindness as only one symptom in a general lowering of mental ability; and

(3) One which attributes the condition to a more specialized lowering of power in the primary visual centres, rendering true visual perception of words and of other complex sense-data difficult.

The main problems raised by these different interpretations are two in number. Is inability to learn to read or the loss of the power of reading due to specific or to general defect? If the former, does the defect show itself only in reading, or does there appear to be any general lowering of visual power? A psychological investigation should throw light on these points.

The subjects mainly used for the investigation were twenty-six in number—all children between the ages of 9 and 16 years, who were in attendance either at ordinary elementary schools (4) or at special schools for mentally defective children (22). They were selected on the report of their teachers as finding reading a very great difficulty. Before the work peculiar to the investigation was begun, all the children were tested (*a*) with the Stanford revision of the Binet scale, in order to get

some idea of their general mental capacity, and (*b*) with various recognized reading tests, in order to get an estimate of their reading power.

The results of these tests show that the subjects differed very considerably in general ability as measured by the Terman scale—the intelligence quotients varying from 50 to 111. Classification on the basis of these results gives the following groups :—

1 subject has superior intelligence	I.Q. = 111
4 subjects belong to the class dull and backward	I.Q. = 82-88
8 subjects show border-line deficiency	I.Q. = 70-79
13 subjects are classifiable as morons of different grades ..	I.Q. = 50-69

Four of the subjects from the first two groups were in normal schools, the fifth in a school for the mentally defective. It is interesting to note that this boy fell below the others of the same group, chiefly in reading ability. He was, however, better at reading than the brightest boy, who showed superior intelligence.

Testing for reading power in particular revealed also a great variation in the ability of the subjects. Some of them could recognize no single letter, word or figure with any certainty; others could read simple words and knew all letters and figures well. It was estimated, however, that no child was less than four years retarded in reading ability and some of them showed retardation much greater than this. Even the children who could read best had a marked difficulty in writing from memory words which they could read—they could not spell.

No relationship existed between the subjects' intelligence quotients and their power in reading. Two of the worst readers were the least intelligent and most intelligent boys. The three worst cases examined, i.e., cases with no reading power at all, had intelligience quotients of 61, 79 and 78 respectively. Many defective boys with such high intelligence quotients read quite well.

The tests in the scale used for finding the intelligence quotients gave no indication of any special disability common among the children. On the whole they failed most on tests requiring auditory repetition, but this characteristic does not mark them off from defective children who can read.

The main suggestion of the preliminary work, then, is that inability to learn to read depends rather on a specific than on a general defect, although in school life such a defect may, because of its nature, simulate one more wide-spreading in character.

Experimental Work.

The experiments aimed at investigating each subject's ability in the various forms of mental activity known to be involved in the complex act of reading, in so far as that act implies the power to recognize words, letters and figures previously taught. Reading proper, i.e., reading new words, involves, of course, yet further powers, in particular the power of ready analysis and synthesis and quickness in applying what is known to new situations; and for this reason probably most defective children are slow in reading and in learning to read. It was, however, in their failure to retain what had actually been taught that our group of non-readers showed their extreme peculiarity, and because of this fact the experiments aimed primarily at testing activities required here. These activities fall into three groups:—

(1) Rapid and easy visual discrimination of forms, together with the ability to retain these without undue repetition.

In particular this discrimination must be extended to forms which closely resemble each other (e.g., ran, ram), and also to those which differ only in the arrangement of their parts, (e.g., b, d, p—god, dog—saw, was—&c.). It may here be noticed in passing that this power of retaining the knowledge of the relationship of the parts to each other and to the whole in a visual percept is one often late in development.

(2) A similar rapid and easy discrimination of sounds.

(3) The possibility of establishing readily an association between a given form and a given sound.

Hence the experiments used can also be arranged in three groups according to the special power which they aimed at testing. Each was carried out with most of the members of the non-reading group, although it was not always possible to include them all. In each experiment the results of the group of non-readers tested are compared with those of a group of readers, equated as far as possible with the non-readers with regard to age and mental ability.

All the experimental work was carried out individually with each child, the procedure being the same throughout the experiment in all cases.

§1.—*Experiments to test the Powers of Visual Discrimination and Retention.*

Experiment 1.—The aim of this experiment was to test the subject's ability to distinguish between unlike and to recognize like forms when these are presented in sequence. In particular it was planned to reveal whether and how far—

(1) Forms showing right and left or top and bottom reversal can be distinguished in memory, and

(2) Forms containing common elements tend to be confused.

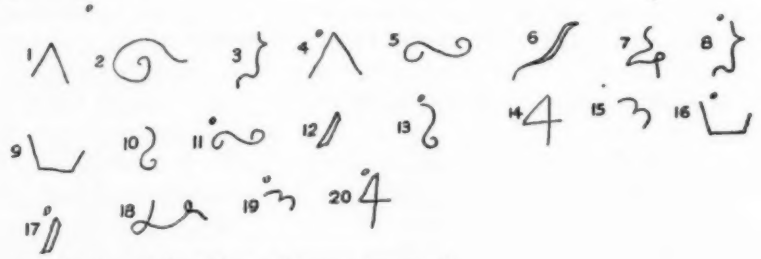
Eighteen series of forms were prepared, each series containing some quite different and some similar or like forms. The number of forms in each series was twenty; of these eight were either exactly like or closely resembled others. The same form was never used in more than one series. Each series was taken as a separate unit and the subjects were told that they were to make their judgments in relation to the forms of that series only.

The forms were shown to the subjects individually, each being exposed for a period of two seconds. After each exposure the subject was required to say "Yes" if he judged the form to be like one already seen in the series, and "No" if he judged it to be a different form.

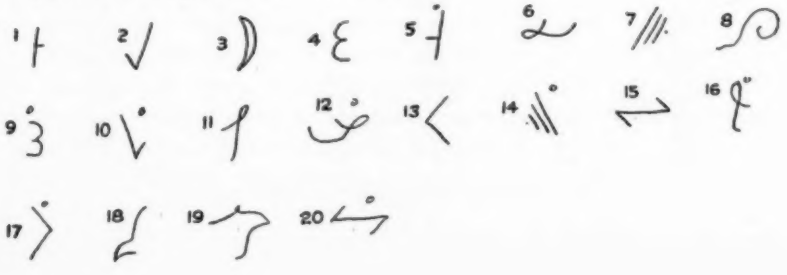
Care was taken to prevent the repeated forms from occupying similar positions in the different series. As a rule, two series of forms only were exposed during one sitting with each subject. In this way, the possibility of confusion with forms of other series was reduced; as a matter of fact there is no evidence to show that such confusion constituted a source of error.

The following are examples of the forms used:—

Series 1.—Repetition of like forms. (° Indicates that the form is a repeated one.)



Series 3.—Repetition of forms inverted.



The repeated forms in the different series were :—

Series 1 and 2. Exactly alike.

Series 3 and 4. Alike except for RL inversion.

Series 5 and 6. Alike except for TB inversion.

(After passing judgment on the foregoing series, the subjects were told that the differences in direction were to count as points of dissimilarity.)

Series 7 and 8. Alike except for TB inversion.

Series 9 and 10. Alike except for RL inversion.

Series 11, 12 and 13. Alike except for either TB or RL inversion.

Series 14, 15 and 16. Alike either wholly or partially.

Series 17 and 18. Alike either wholly or in one part—the whole form consisting of three parts.

The results, given in percentages, are tabulated below. Fourteen non-reading boys worked through the whole eighteen series. Twenty-four non-reading boys and seven reading boys (i.e., boys who could read up to or beyond their intelligence level), also defective, worked with the first two series also.

TABLE I.—SHOWING THE ABILITY OF NON-READING BOYS TO RECOGNIZE LIKE FORMS AS LIKE, AND DIFFERENT FORMS AS DIFFERENT.

No. of series		Percentage of answers "Yes" to forms entirely alike or alike in some particular		Percentage of answers "Yes" to forms entirely different
1	..	82·5	..	5·5
2	..	65·8	..	2·7
3	..	66·6	..	1·6
4	..	56·6	..	5·0
5	..	60·7	..	5·3
6	..	59·8	..	5·3
7	..	47·3	..	6·5
8	..	48·2	..	2·4
9	..	50·0	..	2·9
10	..	45·0	..	6·5
11	..	42·8	..	4.8
12	..	45·5	..	6·5
13	..	55·7	..	7.0
14	..	+ 69·2 and °13·4	..	6·4
15	..	+ 51·9 and °28·0	..	7·7
16	..	+ 32·7 and °26·9	..	4·4
17	..	+ 38·0 and °15·0	..	13·9
18	..	+ 51·9 and °25·0	..	5·1

+ = Whole form repeated. ° = Part repeated.

TABLE II.—SHOWING COMPARATIVE ABILITY OF READERS AND NON-READERS IN RECOGNIZING LIKENESS IN FORMS SUCCESSIVELY PRESENTED.

	Percentage of answers "Yes"	
	(1) To like forms	(2) To unlike forms
Series 1 :—		
Readers ..	57·0 ..	3·5
Non-readers ..	76·0 ..	6·9
Series 2 :—		
Readers ..	43·0 ..	6·0
Non-readers ..	61·0 ..	2·4

These results seem to warrant the following inferences —

(1) All the children examined were able with considerable ease to recognize like and to distinguish unlike forms successively presented, when the forms were like or unlike in all particulars. The non-readers were in no way inferior here to the readers.

(2) The non-readers found forms alike except for differences of orientation very difficult to distinguish, even when special attention was directed to that aspect of the form.

(3) Difficulty was also occasioned by repetition of a part of a form in other surroundings.

It was unfortunate that the other series could not be worked through with the reading group. Reference to a similar experiment, however, performed with a group of younger subjects, suggests that the difficulty shown here by the non-readers in dealing with forms differently orientated is considerably in excess of the normal. The results of this younger group show only 26·4 per cent. and 23·7 per cent. of error in dealing with two like series.

Experiment 2.—This experiment was planned to discover whether the length of exposure of a form made any difference to the children's power to recognize it on a second presentation, and if this did occur to see whether the non-readers in particular suffered from shortening of the exposure.

The subjects were fifteen non-readers and ten readers.

The work was similar to that of Experiment 1, a series resembling in construction Series 1 and 2 of that experiment being used. It differed only in that here, by the use of a simple drop-shutter apparatus, the exposure-time of each form was momentary, instead of being, as in the first experiment, two seconds in length.

TABLE III.—SHOWING COMPARATIVE ABILITY OF READERS AND NON-READERS IN RECOGNIZING LIKE FORMS UNDER CONDITIONS OF MOMENTARY EXPOSURE.

	Percentage of	
	(1) Correct answers	(2) Incorrect answers
Readers ..	45·0 ..	19·0
Non-readers ..	56·0 ..	10·0

There is no evidence here that non-readers as a class fail in speed of perception or in ability to recognize forms with material of this type.

Note.—In spite of previous practice with the apparatus all the children found this work much more difficult than the related work in Experiment 1, and it is not felt that the results are very trustworthy, since certain individuals, finding the work beyond their powers, tended to respond automatically—saying "Yes" or "No" to all the forms. Cases in which this tendency operated throughout have been omitted from the results.

Experiment 3. *Reproduction of forms.*—In order to test further the effect of length of exposure on the ability to perceive a form in the two selected groups of subjects, an experiment was planned which demanded the reproduction of forms seen only with momentary exposure.

The forms were exhibited by means of the apparatus already mentioned, each form being shown three times in succession. After each exposure the subject was required to draw what he had seen. Finally the subjects copied each of the drawings from the original card.

Twenty boys, ten readers and ten non-readers, performed this experiment.

It is in practice impossible to judge the results by any allocation of marks, but the general impression gained by a careful examination of the drawings is that there is nothing to distinguish the work of the non-reading group of boys from that of the readers. Some of both groups reproduce well and some reproduce badly. A much higher correlation exists between the subjects' reproductions and their drawings from the copy than between their reproductions and their ability to read.

We seem, then, justified in inferring that slowness in visual perception is not an important factor in causing difficulty in learning to read.

Experiment 4. *An experiment in learning from visual material.*—The aim of this experiment was to discover whether the boys grouped as non-readers showed any special disability in learning from visual material, the method of learning being left free.

To this end four large squares were prepared, each divided into nine small squares. Each small square was filled with either (1) a colour (all the subjects could name every colour used quite accurately); or (2) a simple geometrical drawing or a representation of some common object.

Each large square was shown to the subject for a fixed period (thirty seconds). He was then required to indicate on a duplicate skeleton square the contents of each small square.

This experiment was performed by thirty-six boys, twenty-four non-readers and twelve readers.

The filled-in duplicate squares were marked as follows:—

(1) Each form or colour correctly placed received one mark; (2) each form or colour correctly given but misplaced by not more than one square in any direction received half a mark.

The marks gained by the respective groups are given below:—

TABLE IV.—RESULTS OF THE COLOUR SQUARE EXPERIMENT.

The figures given indicate the percentage of actual in relation to possible successes with each square, each group of boys being taken as a whole.

Number of square:	1	2	3	4
Readers ..	45	38	43	52
Non-readers ..	44	39	49	42

Examination of these results makes it quite clear that from this experiment no evidence can be obtained which would indicate that non-readers find learning from visual any more difficult than do readers of equal mental attainments. The percentage of successes in the two groups are almost identical.

In all cases, however, the method of learning employed was audito-motor-visual.

Further, success in this experiment did not depend on the power to differentiate between forms nearly alike or forms differing only in position. Results were considered as right if the drawings made were recognizable, irrespective of the accuracy of their details. Nearly 50 per cent. of the errors made were due to misplacement of the colours or forms, while many of the drawings showed faulty orientation.

Experiment 5.—In common with the two succeeding experiments, Experiment 5 aimed at carrying further the investigation already begun in Experiment 4, i.e., it was planned to discover whether the reading and non-reading groups differed in their ability to learn from visual material. These three experiments, however, differed from Experiment 4 in that—

(1) The material used (digits and words) was actually of the kind ordinarily used in reading. Learning from it, therefore, demanded the power of discriminating rapidly between very similar visual presentations.

(2) The actual learning was more definitely visual in type. As far as possible auditory and motor aids to learning were prevented.

The fact that material such as digits and words could be used for experimental purposes with children defined as non-readers requires perhaps some explanation. The tests of reading ability were made at the beginning of the experimental work; these tests were made near its end, some nine months later. In the meantime attempts to teach reading had been carried out side by side with the actual experiments. No child had been found entirely incapable of learning any words or figures when taught alone in this way. At the end of the nine months, therefore, all the subjects used for these three experiments could name all the words and figures employed in them quite well.

In Experiment 5 digits were used as learning material. A typed list of numbers was prepared, having on it four 3-place, four 4-place, and four 5-place numbers. These were exposed in turn to the subject, each for a period amounting to one second for each digit. Immediately after each exposure the subject was required to give the number seen.

The work was done by eleven readers and twenty-six non-readers.

The results were marked in terms of error as follows:—

(*a*) Omission or insertion of a digit, or the substitution of one digit for another = 1 error.

(*b*) Misplacement of a digit by not more than one place = ½ error.

(*c*) Interchange of two neighbouring digits = ½ error.

TABLE V.—SHOWING AVERAGE ERROR PER BOY IN THE REPRODUCTION OF DIGITS LEARNT VISUALLY.

Number of digits in group:	3	4	5	Total
Readers ..	0·23 ..	1·27 ..	3·3 ..	4·8
Non-readers ..	1·9 ..	4·6 ..	8·8 ..	15·3

A glance at the table given above is sufficient to indicate the comparative failure of the non-reading group here. An examination of the nature of their errors is even more instructive. The chief causes of error were:—

(*a*) Misplacing of digits in the number given, and

(*b*) Misreading of digits similar in form, e.g., 6 and 9, 5 and 8, 5 and 3, 3 and 8, &c.

The results of this experiment, therefore, seem to confirm those

found in Experiment 1, viz., that non-readers as a class find difficulty in distinguishing between visual impressions which closely resemble each other, although they are readily able to appreciate and to retain more marked differences.

Experiment 6.—*To test the power of reproducing numbers presented visually with momentary exposure.*

The main aim of this experiment has already been indicated (Experiment 5). Its further object was to test the effect of length of exposure on the reproduction of numbers.

The numbers used were printed on cards and exposed in the drop-shutter apparatus previously mentioned. Five exposures of each number were made, the subject's reply after each presentation being noted. The numbers used were :—

(1) 71	(4) 65	(7) 27408	(10) 914	(13) 3502
(2) 92710	(5) 7930	(8) 049	(11) 36019	
(3) 629	(6) 5186	(9) 4917	(12) 834	

TABLE VI.—SHOWING THE RELATIVE ABILITY OF READING AND NON-READING BOYS IN THE REPRODUCTION OF NUMBERS EXPOSED MOMENTARILY.

Group A.—Twelve Readers.

No. of exposure :	1	2	3	4	5
Card 1	2	0	0	0	0
2	25½	17½	10	7	2½
3	15½	5	2½	2½	3
4	4½	2½	3½	1½	1
5	14½	10½	11	7	5
6	9½	5½	4	2½	3
7	12	8½	2	1	0
8	8½	2	1	1	2
9	27½	14½	11	7	6½
10	2½	2	1	1	1
11	12	7	5	5½	2
12	3	2½	2	2	2
13	15	7½	4	3½	3½
Total	152	85	57	41½	31½
Average error per head	12·6	7·0	4·7	3·4	2·6

Group B.—Ten Non-readers.

No. of exposure :	1	2	3	4	5
Card 1	6½	2	1½	1	0
2	29½	20½	15	13½	7½
3	16	15	11½	15½	10½
4	6½	5	5	2½	2
5	30	23½	13½	13	8½
6	9½	9½	6	4½	5½
7	21½	10	8	5½	4
8	3½	1	0	0	0
9	25½	16½	12½	8½	10
10	9	5	4	3	3
11	4	1	1	0	0
12	14	12	9½	10½	6½
13	13	12½	10½	10	7
Total	187½	133½	98	87½	64½
Average error per head	18·7	13·3	9·8	8·7	6·4

The results were marked as in the preceding experiment. The number of errors on each successive exposure (taking all the digits together) was then found for each group of boys. Finally, the average error per head per exposure was estimated for the two groups.

Table VI gives the actual results.

These results entirely support the inferences made from the preceding experiment, for the inferiority of the non-reading group is evident. It is hard to say, however, how far the comparative failure of this group is due to the difficulty previously noted, their inability to perceive small differences in visual presentations. That this is a difficulty is clear, but that it is the only or even the main difficulty is doubtful. The children's behaviour in this experiment in particular suggested that part of their failure was due, not only to their inability to retain the visual impression, but to the slowness of their association processes, which prevented their getting sufficiently quickly the name of the number presented.

Experiment 7.—*To test the subject's ability to recognize words and letters shown under conditions of momentary exposure.*

The material used in this experiment consisted of the following words and nonsense words: (1) window; (2) char; (3) bok; (4) floor; (5) feg; (6) tadel; (7) flore; (8) cair; (9) pen; (10) bo; (11) wall; (12) box; (13) table.

They were printed separately on cards and exhibited in the drop-shutter apparatus. Three exposures were made of each word, the subject being required to state what he had seen after each exposure.

Nine readers and ten non-readers performed the experiment. The ten non-readers could, however, recognize under ordinary conditions all the letters and all the words used.

The results were marked on a scheme similar to that used in the preceding experiment. Table VII gives the percentage of error in the two groups:—

TABLE VII.

	No. of exposure:	1	2	3
Words and non-words	Readers	35	21	22
	Non-readers	38	23	13
Words alone	Readers	26	16	14
	Non-readers	27	19	10
Non-words alone	Readers	59	34	30
	Non-readers	53	27	18

Contrary to the results of the preceding experiments, no indication is given here of any difference in ability between the reading and

non-reading groups. Two reasons may be offered to explain this fact:—

(1) Since the non-reading children had learnt these particular words and letters, they may, for purposes of this experiment, be considered readers. This argument could, however, be equally applied to the work with digits.

(2) It is well known that very little is actually seen under conditions of momentary exposure—what we do see is added to through association and depends very largely on what we expect. This being so, the group of non-readers was at a considerable advantage in this experiment, for they knew only a few words and expected those words to occur in any reading exercise. The readers, on the other hand, had a much wider range of words called up by what they saw, and so were the more liable to error.

It is now possible to summarize certain general inferences that can be made with regard to the power of non-reading children in visual discrimination and learning. Comparing them with readers of equal mental ability, we find that these children are able:—

(1) To discriminate between unlike forms and to remember them in a normal degree, except in cases in which the forms (*a*) differ only in part or (*b*) are alike, except in their orientation.

(2) To make such discriminations equally with the normal, whatever the length of exposure time of the form.

(3) To learn from visual material also in a normal degree when the method of learning is free and the forms easy to distinguish, but

(4) To find such learning abnormally difficult in cases in which it is (*a*) more exactly visual and (*b*) concerned with easily confused material, e.g., digits.

The main reasons suggested as a cause for this trouble are slow discrimination of similar visual presentations and faulty association between a visual impression and its appropriate name. Probably both causes operate in every case.

§ 2.—*Experiments to test Auditory Discrimination and Retention.*

Experiment 8. *The auditory learning of digits.*—The work of certain of the non-reading subjects had indicated that possibly a specific auditory difficulty existed in some cases of inability to read. The two following experiments were, therefore, planned in order to test the power of the non-reading group as a whole in auditory discrimination and retention. In this first experiment the material used was digits—

in number and arrangement exactly similar to those used in Experiment 5, i.e., four 3-place, four 4-place and four 5-place numbers were employed.

The numbers were read by the experimenter at a uniform rate of one digit per second and immediately after the completion of each a reproduction was attempted by the subject.

The results were marked according to the scheme given in Experiment 5 (p. 293). They are recorded below.

TABLE VIII.—SHOWING AVERAGE ERROR PER BOY IN THE REPRODUCTION OF DIGITS LEARNT BY HEARING.

Number of digits in group:	3	4	5	Total
Readers	0	0·13	2·6	2·73
Non-readers	0·12	0·94	4·44	5·5

The non-readers as a group have greater difficulty in reproducing digits presented auditorily than do the readers, although their difficulty is not relatively so great as with visual presentation of similar material.

The work with digits was carried out by exactly the same boys in both groups—readers and non-readers.

Experiment 9. *The auditory learning of sentences.*—The aim of this experiment was to test the power of learning among the non-readers from auditory material other than digits.

Twenty-four sentences were used, including four with each of the following number of syllables: 10, 11, 12, 13, 14 and 15 syllables. Each sentence was read aloud to the subject and an immediate reproduction required.

The results were then marked according to the following scheme :—

(1) Insertion or omission of any word = 1 error.

(2) Misplacement of a word or phrase = ½ error.

The subjects were twenty-three non-readers. The value of the results was estimated by comparison with what is normal for the mental ages of the children concerned.

The actual numerical results (in terms of error) are given in Table IX (page 299).

Most children of from 6 to 7 years of age are able to reproduce accurately sentences of from sixteen to eighteen syllables. Almost all these subjects had a mental age of over 7 years—a fact which indicates that their difficulties in this experiment were far greater than would be expected.

These results, therefore, are in agreement with those of the preceding test—the non-readers as a group show a special disability in retaining and reproducing a series of auditory impressions.

TABLE IX.—SHOWING THE NUMBER OF ERRORS MADE BY EACH SUBJECT IN THE REPRODUCTION OF SENTENCES.

Number of syllables in each sentence		10	..	11	..	12	..	13	..	14	..	15
Total number of syllables to be reproduced in group of four sentences		40	..	44	..	48	..	52	..	56	..	60
Errors made by subject	(1)	3	..	2	..	15	..	7	..	7½	..	22
	(2)	0	..	0	..	0	..	2	..	2	..	5
	(3)	2½	..	3	..	5	..	6	..	11	..	26
	(4)	0	..	1	..	8	..	3	..	10	..	19
	(5)	0	..	2	..	2	..	2	..	7	..	15
	(6)	1½	..	1	..	10½	..	5	..	15½	..	34
	(7)	2	..	1	..	3	..	6	..	11	..	16
	(8)	2	..	5	..	8	..	3	..	18	..	25
	(9)	0	..	0	..	0	..	1	..	6	..	11
	(10)	0	..	0	..	0	..	4	..	0	..	3
	(11)	0	..	0	..	0	..	0	..	1	..	0
	(12)	0	..	0	..	0	..	0	..	0	..	0
	(13)	0	..	1	..	2	..	1	..	1½	..	2
	(14)	0	..	0	..	1	..	2	..	5	..	7
	(15)	0	..	0	..	4	..	2	..	0	..	5
	(16)	0	..	0	..	0	..	0	..	0	..	2
	(17)	2	..	0	..	3	..	0	..	1	..	9
	(18)	5½	..	3	..	14	..	14	..	19	..	32
	(19)	0	..	0	..	0	..	1	..	1	..	2
	(20)	1	..	0	..	0	..	0	..	6½	..	6
	(21)	0	..	0	..	2½	..	3	..	6	..	10
	(22)	0	..	0	..	0	..	0	..	1	..	2
	(23)	1½	..	9	..	19½	..	11	..	18½	..	39

Certain points are, however, of special interest:—

(1) Individual differences in capacity revealed by this experiment are very great, much greater than those shown when visual material was used. These differences show no correlation with variations in general mental ability.

(2) Certain subjects whose auditory power as measured by these experiments is very limited, are among the worst readers in spite of the fact that their general mental capacity is comparatively high.

(3) No correlation exists between the findings in the auditory tests and those of certain visual tests, e.g., Experiment 1: correlation between results of Experiments 1 and 8 = ·073, between Experiments 1 and 9 = −·21.

With regard, then, to the auditory capacity of the non-reading group, we may say:—

(1) That the non-readers as a group show a lowering of auditory power, but that their difficulties in dealing with auditory material are not, on the whole, so great as their difficulties with visual material.

(2) No correlation exists between auditory power as measured by the tests and certain forms of visual power. Defect in either capacity

seems to be specific rather than an expression of a general lowering of ability.

(3) That certain individuals show auditory difficulties to a peculiarly marked degree. These individuals are especially bad in reading.

§3.—*Experiments to test the Power of making Audito-Visual Associations.*

We have now to consider the experiments in the final group.

Experiment 10.—The aim of this experiment was to try to estimate the subject's ability to form an association between :—

(*a*) A given form exhibited visually ; and

(*b*) A common name.

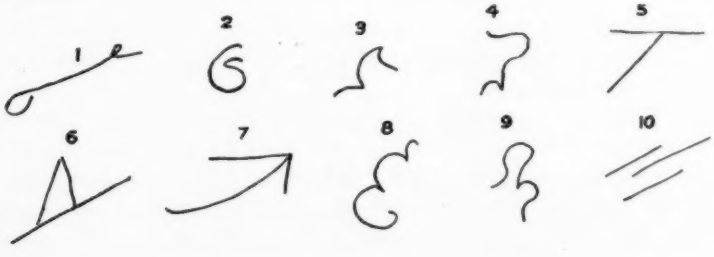
Three sets of forms were prepared—ten in each set—somewhat similar to the forms used in Experiment 1, except that in this case there were no repeated forms. Corresponding to each set of forms a list of ten common names was made.

The subjects were shown the forms each in turn for a period of two seconds—the experimenter in the meantime saying, and the subject repeating, the associated name. Immediately after the last form had been shown, the forms were shown again in an order different from that originally used, and the subjects were required to give their associated names. If the subject failed to give the name after a period of two seconds, it was again given by the experimenter and repeated by the subject. The forms correctly named were put on one side and not given again. This procedure was continued until all the names were known.

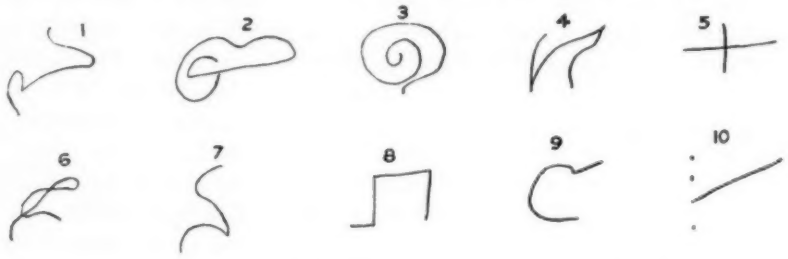
Illustrations of the forms are given below.

Series 1. *Names :* (1) dog ; (2) potato ; (3) waggon ; (4) frog ; (5) train ; (6) grass ; (7) chair ; (8) coat ; (9) moon ; (10) window.

Forms :



Series 2. *Names:* (1) shoe; (2) mud; (3) sun; (4) boat; (5) road; (6) tree; (7) post; (8) button; (9) sheep; (10) pond.



The experiment was carried out by twenty-six readers and thirteen non-readers, and the result is shown in Table X.

TABLE X.—SHOWING THE PERCENTAGE OF FORMS CORRECTLY NAMED AT EACH SUCCESSIVE EXPOSURE, THE RESULTS OF (*a*) ALL THE READERS AND (*b*) ALL THE NON-READERS BEING TAKEN TOGETHER.

	Series 1						Series 2					
Exposure	1	2	3	4	5	6	1	2	3	4	5	6
Readers	59	18	11	6·9	3	—	46	20	17	6	3	—
Non-readers	50	20	18	7	2	—	48	23	10	10	2	2

Clearly there is from this experiment no evidence to indicate that the non-readers find association of this type any more difficult than do the readers. In fact, two of the worst readers (boys unable to read any words and in one case any letters or figures) make two of the best scores, whilst the worst score but one is made by a boy who reads fluently.

Certain subsidiary points are important, viz.: (1) There is no evidence to show that the place occupied by the form in the original series has any influence on the ease of recall of the associated name; rather (2) the relative ease of reproduction of the associated name seems determined to a large degree by the nature of the form. This is well shown in Table XI.

TABLE XI.—SHOWING THE RELATION BETWEEN—

(*a*) The nature of the form, and
(*b*) The number of successes gained on the first attempt at naming.
(All the subjects taken together.)

Series 1—										
Number of form ..	1	2	3	4	5	6	7	8	9	10
Number of successes on first naming ..	17	27	9	16	27	23	28	22	10	28
Series 2—										
Number of successes on first naming ..	12	14	18	9	22	7	6	20	12	16

The fact that the possibility of remembering the associated name depended, at least in part, on the nature of the form is a particularly interesting one. That it did so depend seems clearly indicated by the results. Forms 7, 10, and 5 of Series 1, and Forms 5 and 8 of Series 2 could not otherwise have shown to so great an advantage. The names were all ordinary names and it is unlikely that one or two of them should have meant so much more than the rest to a number of children with widely differing environments as to account for the results shown. No child was in the least unfamiliar with any name and the names were remembered almost always when the association was forgotten.

It is difficult to draw any definite conclusions as to what form characteristics rendered naming easy; but it certainly appears as if one important factor was the shape of the forms concerned. A form which could be mentally described by the subjects seemed to be most easily named. Those worst named (Series 1 to 3, Series 2 to 6 and 7) were all of an indefinite shape, not readily to be described in words.

And the accounts of how they remembered given by the subjects lend colour to this view. "There were three lines." "It had dots and a line." "It was round," &c.

The most successful associations, then, were not simple direct associations between a given visual impression and a given sound. They were associations between a visual impression which had meaning —which was consciously analysed and its parts related definitely to each other—and a sound also full of meaning. For even more often in the records is there indication that the association depended for its retention on the meaning of the word as well as on that of the form, e.g., Series 1 to 2 is said to be remembered "because a potato is round"; Series 2 to 5 "is like a road"—"it is straight"—"it is a cross —cross-roads," while No. 8 of the same series is known "because it's nothing like a button," "a button isn't that shape," &c.

Suggestive, too, is the behaviour of the subjects. They all enjoyed the experiment and many of them were amused by it. But the attitude of any individual in accepting the associated names for the first time differed very considerably, apparently depending on whether he did or did not consider that the name fitted the form. To some forms the attitude was one of doubt or question, as if the name had not quite been heard; in the case of others there was a ready acceptance of the name and a look of pleasure. "Button" frequently induced laughter.

This difference, i.e., the fact that the nature of a form, either

because of its shape or because it fits itself readily on to the associated sound or the meaning of that sound, determines the ease with which the associated sound is retained—is, of course, nothing more than is generally found with children in learning letters. Therefore, some letters, (e.g., o), are particularly easy while others are hard.

Summarizing the conclusions that can be drawn from these facts it appears :—

(1) That in forming an association between a visual presentation and a name, the element of meaning is of primary importance.

(2) That this meaning factor may be inherent in the form itself, or may depend on the nature of the form considered in relation to the meaning involved in the name.

(3) That the less distinctive the forms are in themselves the less easy also is the retention of their associated names; and that therefore

(4) Any difficulty which leads to failure to grasp fully the details of presented forms or to inability to distinguish between them will be likely to render verbal associations with them difficult also.

Experiment 11. *The association of words and Greek letters.*—This experiment aimed at testing the subject's capacity to form associations between common names and letter forms, i.e., it aimed at testing the hypothesis formulated from the results of the last experiment, that the meaning factor inherent in a form is an essential factor in establishing verbal associations with that form.

The material used resembled that used in Experiment 10, except that Greek letters were substituted for the more distinctive forms. The method of procedure was also the same as before.

The letters used were (1) γ; (2) δ; (3) η; (4) λ; (5) μ; (6) ν; (7) ξ; (8) ψ; and the names (1) cow; (2) river; (3) flower; (4) box; (5) wall; (6) paper; (7) rain; (8) hen.

The results show that here the associations were harder to make than in the preceding experiment. In spite of the fact that only eight letters were used as against ten forms, the percentage of successful responses on the second showing of the letters was considerably less than it had been in Experiment 10, i.e., 41 per cent. as against 51 per cent.

The subjects themselves said that the work was harder because the forms were "like each other." There was a constant tendency to confuse "cow," and "box" and "cow" and "paper," owing to the similarity of the forms γ and λ and γ and ν.

It was more difficult in this experiment than in the former to get

any indication from the subjects themselves as to what memory depended on. Usually they declared that they did not know how they remembered. There is, however, some evidence of the influence of meaning. γ was said to be "like horns," ξ to "look like rain," &c.

We find then that the results of this experiment seem to give support to the conclusions previously drawn. They show clearly that it is more difficult to form associations between common names and forms which closely resemble each other, not having in themselves any definite characteristics, than between similar names and more distinctive forms.

Experiment 12. *Learning the names of eight Greek letters.*—An attempt was now made to estimate the subject's power to establish a connection between eight Greek letters and their proper names. The procedure was identical with that of the preceding experiments.

The letters used were α, β, σ, ρ, ζ, ϕ, π, θ.

There is great difficulty in estimating the value of the results in this case, for various reasons, viz. :—

(*a*) Often the subject was quite unable to say the name after it had been said by the experimenter. Consequently it had to be repeated—sometimes more than once. This fact meant that the time of the original exposure in this experiment was much longer than in the two preceding ones, and a repetition factor, not present with them, was involved.

(*b*) When the answers (i.e., the associated names) were being given by the subjects, it was often impossible to tell whether the right answer was being given or not. This was particularly so with many of the boys, who could not, or did not, make correctly such sounds as "th," &c.

(*c*) A considerable tendency to automatism was shown, i.e., one sound (possibly a letter name) was given in answer to all the letters shown. This tendency was due evidently to the felt difficulty of the task. It provides an answer without effort when too great an effort is demanded.

Bearing in mind these facts, with the accompanying result that the children's efforts were judged too leniently, it is interesting to see that here the number of successful responses to the second exhibition of the letters is reduced from 41 per cent. in Experiment 11 to no more than 17 per cent.

A marked difference, also, was shown in the attitude of the subjects. There was little enjoyment. The boys looked worried and baffled, as

if they were up against a task which they could not accomplish. It was often hard to retain their attention. The tendency to automatism has already been noted. Another tendency that was very prevalent was the changing of the name of the letter to one somewhat similar which had meaning to the subject. For example, ρ became "road," ϕ became "fine," and σ was called "signal." The similarity of many of the names led to great confusion; β, θ, ζ were continually muddled, all of them being called indiscriminately "beta" or "meta."

It is interesting to note that 44 per cent. of the successes on the second exposure of the letters were with π, and 32 per cent. with β, which, as one subject justly remarked, "was like B." No correlation exists between the boys' success in this experiment, and in Experiment 10 ($r = 0{\cdot}05$), but a fairly high correlation is shown between its results and those of the experiment testing the power of immediate reproduction of digits presented auditorily ($r = 0{\cdot}63$; $PE = 0{\cdot}16$).

Since, therefore, the only point in which this experiment differed from the preceding one lay in the nature of the name or sound, it is evident that the ease of association between a visual presentation and a sound depends partly on the nature of the sound. The important points with regard to the sound which determine success in reproduction seem to be:—

(*a*) That it shall have meaning.

(*b*) That it shall be readily distinguishable.

Summarizing the conclusions that can be drawn from the three allied association experiments, we find:—

(1) That the non-readers as a group make associations between meaningful material (i.e., dissimilar and characteristic forms and words with meaning) as readily as do the readers; but that their difficulty is increased at a rate in excess of that of the readers with the increase in similarity between the forms and sounds presented.

(2) That in all cases the ease with which an association is established between a given form presented visually and a given name depends on the nature of both name and form, and

(3) That the two points of importance with regard to both form and sound in making for ease of association are: (*a*) that they shall be readily distinguished from each other; (*b*) that they shall have meaning, apart from the meaning inherent in their actual presentation.

It is, of course, easy to see that these two points are but one aspect

of the same thing. The form [is easier to distinguish from other forms and also to remember than the form ; because by its very nature it has more meaning: "It has three lines and two angles"; "It is two horizontal lines joined by a vertical line on the left"; "It is a square with one side left out"; "It is nearly a capital E," &c. In the same way "cow" has more meaning than "beta," although in this case the meaning is not inherent in the form of the word.

Hence cow and [are more easily retained than and beta.

Further, and button can be associated because "is not like a button," and γ can be associated with cow because "a cow has horns"; whereas between and button, and between and beta no links of attachment are likely to occur to the mind.

In the same way, when the figure 6 is known, the letter b may be successfully learnt in spite of years of failure if it is realized that the "bee has six legs."

We see, therefore, that any difficulty which hinders a rapid grasp and differentiation of forms and sounds will be likely to hinder their retention and their association, since the hooks of memory will in such a case be either absent or few in number.

Summary.

It is now possible to estimate the bearing of the experimental work which has just been described on the theories commonly held as explanatory of the condition having as its most obvious symptom failure to read or to learn to read. Three points are important.

(1) It is clear from the facts (*a*) that non-readers are found among all degrees of intelligence; and (*b*) that the degree of failure in reading, at least among normals and high-grade defectives, shows little correlation with the degree of "general defect" commonly so called, that the defect underlying inability to read is to a certain degree specific in nature. The idea that deficiency in reading power is always an indication of a deficiency of a more general kind is, therefore, by no means supported by the psychological facts; although, of course, the condition known as "general defect" is more frequently accom-

panied by loss of power in this as in all other abilities than is normal mentality.

(2) Yet there is nothing in the results of the experiments to indicate the existence of any such region as a "visual-word" centre, the absence of or injury to which will make the visual recognition of words impossible. The defects found are not so strictly localized as such a hypothesis would demand, for the word-blind individuals reveal special difficulties in dealing with material other than words. Further, the implication of this theory that ability to read depends on the power to store up images of words has no psychological support; the recall of images is not in question.

(3) The theory that the experiments do support is that "word-blindness" is but one aspect of a more general, yet still in itself specific, defect in either the visual or auditory regions or in both. All the non-readers examined showed a reduction of the normal power in dealing with forms visually presented—especially when these forms were very like each other, their defect being shown most definitely in their failure to remember such forms. Further, certain of the non-readers showed corresponding defects in the auditory region—they could not readily discriminate or retain similar sounds. Some of the worst cases had defects of both kinds. Taking the group of non-readers as a whole, however, no correlation could be found between auditory and visual ability, i.e., these defects appeared also to be specific, although occasionally found together in one subject.

Certain of the experiments indicate that the cause of the failure to associate, as well as to retain, sounds and forms, lies to some degree in this primary disability of the auditory or visual regions, resulting as it does in the failure of the forms or sounds presented to gain any meaning. Whether there is also a failure in primary retention, declaring itself in the failure to form memory images to a normal degree, is not known. An inquiry into the use of imagery, depending as it does on the introspection of the subject, is difficult with defective children. Many of them used visual imagery in other matters, but they may, nevertheless, have been unable to visualize forms which had no meaning for them.

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A PROPRIOCEPTIVE REFLEX AND CLONUS AS STUDIED IN THE SPINAL FROG.

BY KANSHI SASSA.

(From the Physiological Laboratory, Oxford.)

IT is somewhat remarkable that although the frog so commonly has furnished the material for laboratory observations on muscular and lower nervous reactions, and although the experimental work upon the knee-jerk and other tendon reflexes has been so extensive, the literature of this latter subject contains, so far as I can find, but one reference to tendon reflexes in the frog. That reference is by Langelaan [3] (1901), who mentions the knee-jerk as being obtainable in a lively frog. I imagine that the reason for the general absence of reference to these reflexes in the frog is that their elicitability in that animal is quite exceptional. Rollett [5] reported that no reflex is evoked from the sterno-radialis tendon of the spinal frog in any way in spite of the existence in it of abundant nerve organs. I have myself never obtained unequivocal evidence of the knee-jerk in the frog. I find, however, that there is a reflex of the proprioceptive kind quite commonly elicitable in the spinal frog, and especially well marked in the autumn and early winter, when the animals are more excitable reflexly and in good condition. This is a proprioceptive reflex not of an extensor but of a flexor muscle, semitendinosus, a flexor of the knee. In the decapitated preparation a quite slight pull on the tendon of semitendinosus evokes a well-marked contraction of the muscle.

I have taken graphic records of this reaction in the following way. The isolated tendon of semitendinosus was carefully prepared and all other tendons inserted round the knee-joint were detached. The semitendinosus tendon was attached by a thread to the vertical short-arm of a light crank lever myograph. To elicit the reflex the horizontal long-arm of the lever, carrying the writing point, was lifted by the finger so as to slacken the muscle. Then, on removing this support, the fall of the axially weighted lever gave a sudden pull to the muscle and this slight pull evoked the reflex contraction. The tension of the pull amounted only to a few grammes. Section of the roots of the sciatic

nerve immediately annulled the reaction. A slight tap upon the thread stretching the semitendinosus was invariably followed in the spinal preparation by the contraction of the muscle. No reflex contractions could be elicited by similar means either in the quadriceps femoris or in the gastrocnemius. Nor in these latter muscles (extensors) was there evidence of tone, but in spinal semitendinosus there was plenty of tone, especially in the autumn and early winter specimens. My experiments were made during the winter months of 1920.

The reflex contraction elicited by the sudden slight stretch resembled in the form of its myogram a single twitch (fig. 2). Its myogram was strikingly different in course and duration from that of the reflex contraction evoked in the same muscle by a single moderate break-shock applied to an ipsilateral afferent nerve; the latter was far stronger and longer (fig. 1). The contraction of the simple proprioceptive reflex

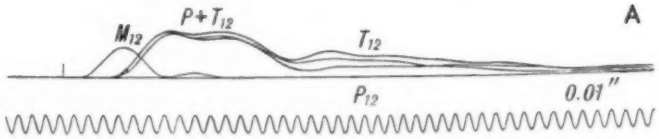

FIG. 1.—Reflex contractions of semitendinosus by single maximal break-shock. Stimuli applied to tibial (T), peroneal (P), and both nerves together (P and T). M is the direct maximal muscle twitch by stimulation of divided roots of sciatic. Numbers indicate distance between primary and secondary coils.

occupied about 0·25 second, which is little longer than that of the simple twitch at the same temperature and season. Thus in one comparison the proprioceptive reflex contraction of the muscle lasted 0·25 to 0·27 second, and the motor twitch of the muscle to a break-shock lasted 0·23 second. The whole form of the curve was very similar in the two. In this respect it seems to resemble another reflex of proprioceptive kind, namely, the knee-jerk. Jolly [2] and recently W. Salomonson [6] have examined the electrical variation of the knee-jerk, and report that the contraction of the muscle—extensor in their case—is accompanied by a simple diphasic variation. As to the latency of the reflex, owing to the mode of excitation not more can be said than that it was not longer than 0·04 second.

When the thread tied to the tendon was gently pulled by the fingers it could be noticed that the reflex response was frequently not one contraction but a short series of successive contractions. The same result could be obtained by increasing the height from which the lever fell for eliciting the reflex, thus increasing the momentum employed in

the stimulus (fig. 2). The reflex response was then a short rhythmic series, each succeeding one smaller than that just preceding it. Sometimes this multiple form of the reflex passes over into a long, lasting clonus. In the graphic records of this clonus the first contractions were of greater amplitude and a little slower period than were the rest, which

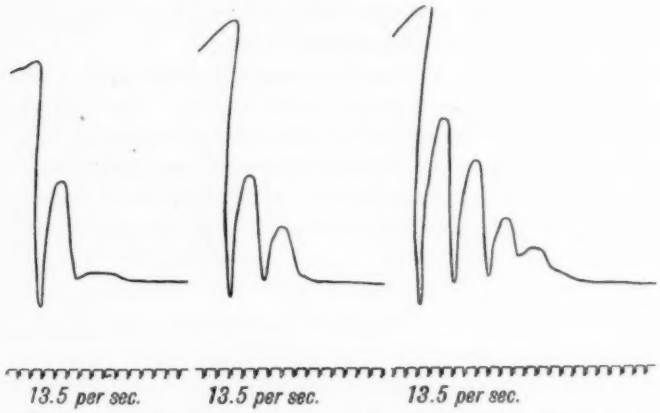

FIG. 2.—Three stretch reflexes from a semitendinosus preparation (showing increase in response with increase of stimulus).

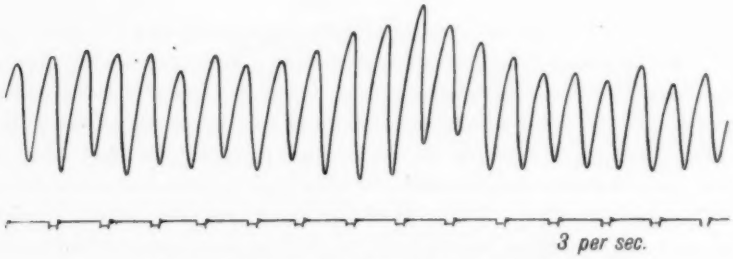

FIG. 3.—Clonus in the semitendinosus of the frog.

were often almost as regular as those of a tuning-fork. The rate varied between 4 to 6 a second; its average was 4·5 a second (fig. 3). The clonus lasted in some cases even for a couple of minutes. It would seem, therefore, that when the momentum of the falling stimulus is suitably adjusted so as to give a repetition of the required tension at suitable speed, the reflex response is elicitable in immediate succession over and over again. But the rate of this repetition is slower in the frog than

in the vastocrureus (knee-extensor) of the cat (Viets [8]), 12 to 17 per second. It is also slower than the ankle clonus (gastrocnemius) of man, which varies between 8 to 10 per second (Waller [9]), and 13·5 to 14 per second (MacWilliam [4]). This proprioceptive stretch-reflex obtainable from the semitendinosus of the spinal frog clearly resembles the reflex of similar type studied in this laboratory by Asayama [1] in the tibialis anticus of the cat. The readiness and relative regularity with which the proprioceptive stretch-reflex is, as the observations show, obtainable from a hind-limb flexor muscle of the spinal frog, but not from the hind-limb extensor muscles, may be connected with the distribution of the postural spinal tone in the two sets of muscles, this tone being in the spinal frog, as Sherrington [7] has pointed out, marked in the flexors but very low in the extensors, in accord with the habitual posture, squatting. The elicitability of the proprioceptive reflex seemed to be good when the spinal tone was well developed.

Conclusions.

A slight quick pull on the tendon of the knee flexor, semitendinosus, in the spinal frog evokes a twitch-like reflex contraction.

This is a reflex contraction, but it is strikingly different in course and duration from the reflex contraction of the same muscle obtained in response to a single break-shock applied to an afferent nerve.

Often this reflex is multiple, i.e., repeats itself under a suitable stretch stimulus. This multiple form of the reflex sometimes passes over into a clonus of long continuance and a rate of about 4·5 per second. This proprioceptive reflex appears to be favoured by the reflex postural tone which in the spinal frog is present in the semitendinosus but not in the extensors (quadriceps, gastrocnemius); from these latter no stretch reflex or clonus was found elicitable.

REFERENCES.

[1] Asayama, C. *Quart. Journ. Exper. Physiol.*, 1916, **9**, 265.
[2] Jolly, W. A. *Quart. Journ. Exper. Physiol.*, 1911, **4**, 67.
[3] Langelaan, J. W. *Arch. f. Physiol.*, 1905, **107**, 94.
[4] MacWilliam, A. *Brit. Med. Journ.*, 1901, **2**, 1595.
[5] Rollet, A. *Sitzungsber. d. Kaiserl. Akad. d. Wissensch. Wien*, 1876, **70**, III Abt., 34.
[6] Salomonson, J. Wertheim. *Brain*, 1920, **43**, 369.
[7] Sherrington, C. S. *Brain*, 1915, **38**, 191.
[8] Viets, H. *Brain*, 1920, **43**, 269.
[9] Waller, A. *Brain*, 1880-81, **3**, 179.

Proceedings of the Section of Neurology of the Royal Society of Medicine.

President—Percy Sargent, C.M.G., D.S.O., F.R.C.S.

Meeting held October 13, 1921.

Some Observations on Epilepsy.

PRESIDENTIAL ADDRESS

DELIVERED BY

Percy Sargent, C.M.G., D.S.O., F.R.C.S.

I feel that I cannot adequately express my appreciation of the honour of being elected to a chair which in the past has been occupied not only by many distinguished men of science, but also by some whose names are indelibly imprinted upon the history of medicine. That a surgeon should be chosen to fill this position is something more than a personal honour; it is a compliment to that branch of practice in which he is engaged, implying as it does a recognition of the fact that surgery has come to occupy a not unimportant place in neurology.

In those affections of the nervous system to which surgical measures are applicable, the physician is chiefly concerned with diagnosis, whilst the surgeon must pay particular attention to the technical problems which operative treatment presents. Neither is fitted by training or experience to perform in these respects the functions of the other, and I do not doubt that if a combined physician-surgeon could be evolved, he would evince the sterility common to hybrids.

I take the opportunity of touching upon this subject in view of the well-known opinion held by Harvey Cushing that the surgeon who concerns himself with operating upon the nervous system should be his own neurologist. However attractive this may be in theory, it can never, to my mind, attain practical realization, for just as a wide knowledge of general medicine is essential to the neurologist, so is an intimate acquaintance with general surgery desirable for one who

takes seriously that part of general surgery which concerns itself with the nervous system. The acquisition of this familiarity with all branches of surgery leaves but scant time for the laborious and exacting work, both at the bedside and in the laboratory, which the training of a neurologist demands. I hold that the nervous system is not a suitable field for surgical specialism in the more restricted sense of the term.

The co-operation of neurologist and surgeon is not only of value in the diagnosis and treatment of certain diseases of the nervous system, but it also affords opportunities for the study of what Moynihan has called the Pathology of the Living. Neurology has already gained much from observations made in the operating theatre, and far more may be confidently expected in the future. The mere exposure of the cortex, the removal of tumours, section of nerve roots and other manipulations reveal phenomena which can be observed neither at the bedside nor by post-mortem examination. In more senses than one the operating theatre may be made a link between the ward and the post-mortem room. When a physician fails to witness the operations which are performed upon his patients, he not only neglects a fruitful source of information for his own future guidance, but he also deprives the surgeon of the very great advantages which accrue from consultation upon questions which may arise during the course of an operation.

In his Presidential Address to the Neurological Society in 1902, Herbert Page said: "I venture to take up the position that surgery may be even bolder than it has hitherto been in endeavouring to minimize and remove some of the causes which seem to be at the root of the later consequences of severe head injury." Among these later consequences the occurrence of epileptiform seizures stands out prominently, more so than ever before by reason of the large number of sufferers from epilepsy resulting from cranio-cerebral wounds.

Having had to deal with many such patients during the past three or four years, I have been increasingly impressed by the feeling that surgical procedures for the treatment of epilepsy are greatly hampered by the obscurity which still exists as to the essential factors that underlie convulsive attacks in general.

James Taylor has recently reminded us that in regarding epilepsy as a disease, there is some danger of losing sight of its purely symptomatic aspect, and has emphasized the desirability of searching in every case for the physical basis which forms the starting point of the convulsive seizures. He contends that the attempt to enmesh epilepsy in the net of psycho-analysis represents a definitely retrograde move-

ment, whilst any real step forward in the elucidation of the nature of "ordinary epilepsy" is to be made from a study of Jacksonian attacks.

Gowers said that a tumour or spicule of bone could "so train the whole brain into a habit of discharge that the attacks differ little from those of idiopathic epilepsy." Instead, however, of merely seeking to reconcile the convulsive seizures of known focal origin with those of idiopathic epilepsy, we should rather approach the problem from the opposite point of view, realizing that in idiopathic epilepsy we have phenomena unassociated with any obvious cause, which may yet be in all respects identical with those which occur in connexion with gross lesions, from the damp squib effort of *petit mal* to the full pyrotechnic display of a major attack. Further, we must remember that between ordinary epileptic fits and focal fits associated with visible lesions there exists a bridge in the form of attacks of a Jacksonian character in which no gross lesion appears to exist.

Surgery would be in a better position to play its part in the treatment of epilepsy, whether or not of focal origin, if we knew more of the factors underlying the phenomena of a fit; and if we could recognize any essential precipitating cause common to all forms.

I would not presume to criticize the various views as to the pathogenesis of epilepsy which have been advanced in the past, or which are at present entertained. I should like, however, very briefly to examine what is known as the vascular hypothesis, because during the frequent opportunities which I have had of viewing the living brain, under both normal and abnormal conditions, I have been greatly impressed by the striking rapidity with which visible circulatory changes may occur in response to various disturbing causes. I cannot help feeling that sufficient importance is not attached to the possible effects upon cerebral function of what may perhaps be called minor and transitory circulatory disturbances. It is not upon the congestion or anæmia of the brain as a whole that I would lay stress, but upon the local variations and abnormalities which can be observed in connection with tumours and various traumatic lesions. It seems to me that the only local influence which an operation can have upon an epileptogenous area, short of excising that portion of the cortex, must be exercised through the medium of the local circulation. If there is a fundamental precipitating cause for all convulsive attacks, any hypothesis which does not take into account the pathogenesis of fits originating in the neighbourhood of a gross lesion must fail to touch that essential point.

Gowers, discussing the pathology of idiopathic epilepsy in 1900,

wrote: "The conceptions of epilepsy which have been current during the last forty or fifty years . . . are not much more instructive than the old demoniacal pathology which gave the disease its name." One such conception he specified, namely, that "the epileptic fit was the result of a spasm of the arteries of the brain," and went on to remark that this notion had disappeared absolutely before the simple but conclusive facts which connected the manifestations of the disease with a morbid state of the cortex. It is noteworthy, however, that, some years later, in the "Borderland of Epilepsy," he admitted the possibility of cerebral vaso-constriction in relation to vaso-vagal attacks. But the view that the phenomena of epilepsy result from disordered cortical function does not by any means necessitate relinquishing all belief in an underlying vascular disturbance.

On the contrary, both the "morbid state of the cortex" postulated by Gowers, and the "spasm of the arteries of the brain" denied by him, may conceivably be concerned together.

The whole subject of the relation of disorders of the cerebral circulation to convulsive attacks, as well as to loss of consciousness and other clinical manifestations, was fully discussed by A. E. Russell in his Goulstonian Lectures in 1909. To these lectures I have long been indebted for suggestions and references bearing upon the very interesting and important question of the relationship between gross cerebral lesions and epilepsy. For it is obvious that in operating upon epileptics, and in choosing cases for operation, we should like to have a clear idea of the purpose of the operation, based upon some reasonable conception of that relationship. The surgeon who merely removes a disc of bone from the cranium of an epileptic patient places himself on a level with the practitioner of the Stone Age, the marks of whose handiwork upon the skulls, probably of the epileptic or the insane, are to be seen in museums of ethnology.

The thesis which Russell developed from a mass of clinical evidence was that the common factor underlying many disturbances of cerebral function is to be found in some disorder of the cerebral circulation. It is interesting to note, in view of the trend of recent research, that as long as twelve years ago he hinted that, so far as the causation of epilepsy at least is concerned, inquiry into the processes, both nervous and chemical, by which the cardiovascular apparatus is governed, might afford a valuable clue. He further drew attention to a similarity between experimental decerebrate rigidity and the tonic spasms occasionally observed in cases of sudden complete arrest of the cerebral circulation.

In most writings upon epilepsy such expressions as "sudden discharge of nerve force," "sudden liberation of energy," and "explosion of nervous energy" are commonly found. Such terms, if they relate to the cortex alone, can only mean that the fit is to be looked upon as a manifestation of excessive cortical activity. It is impossible, however, to regard loss of consciousness, one of the most sudden and striking events, and often the only recognizable event in an epileptic fit, as a manifestation of the discharge of nervous energy.

A sudden failure of the cerebral circulation is inevitably followed by loss of consciousness, and we know that it can also produce convulsive movements, as seen in the attacks associated with the condition known as "heart block." It is inconceivable that a sudden arrest of the cortical circulation can, at one and the same time, bring about both a suppression and an augmentation of function. When loss of consciousness and convulsive movements result from the same cause, we can only believe that those movements point to a suspension and not a stimulation of cortical activity. The tonic stage of muscular contraction appears to indicate, not a discharge of nervous energy from the cortex, but the liberation of a lower level motor mechanism from cortical control. In a word, the unconscious rigid epileptic is, for the moment, a decerebrate man.

In a recent paper Kinnier Wilson has brought forward clinical evidence to show that decerebrate rigidity, both with and without the occurrence of tonic fits, occurs in man as the result of lesions which interrupt the cortico-spinal path, and that such rigidity is strictly comparable with that which obtains in animals when the mesencephalon has been transected. He points out that lesions which have not the completeness of the experimental section find expression in rigidities of limited scope, and that even functional interruption of cortical control may be manifested by a similar sequence of events. It requires no great effort of imagination to suppose that cortical control may be suddenly and momentarily cut off by a sudden and transient disorder of the cortical circulation, and cause, according to the extent and duration of the circulatory disturbance, varying degrees of decerebrate rigidity.

Whilst a state of momentary decerebration may be held to explain the tonic stage of an epileptic fit, it is not so easy to account for the clonic movements. Kinnier Wilson, in the paper alluded to, having characterized the tonic fits observed in the cases which he was describing as "attacks of decerebration," remarks that the absence of

any clonic movement is of much significance. He takes it to indicate that tonic and clonic movements have different origins, and are the expression of the activity of different motor mechanism, the one being characterized by static, and the other by phasic activity.

In a few recorded cases an epileptic fit has occurred during actual examination of the heart or radial pulse. The tonic stage has coincided with the sudden arrest of the circulation, whilst the clonic stage has commenced with the return of the circulation. In cases such as Russell's where sudden and permanent arrest of the circulation was immediately followed by tonic spasm, no clonic movements were observed.

It can hardly be doubted that the clonic movements which follow the tonic stage of a general epileptic fit are of the same character and depend upon the same motor mechanism as those of a Jacksonian fit which is accompanied neither by a tonic stage nor by an initial loss of consciousness. They exhibit the phasic character of movements associated with cortical activity, and resemble both those which can be evoked by faradic stimulation of the cortex, and those of a Jacksonian fit dependent upon a cortical lesion.

Many years ago, Long Fox, who attributed the tonic spasm of an epileptic fit to cerebral vaso-constriction, ascribed the clonic movements to a "gradual yielding of the vasomotor constriction, allowing at first more blood to enter the arteries than during the period of tonic spasm, but yet far less than is necessary for controlled movement or for rest."

That the cerebral circulation is under direct vasomotor control was long denied, chiefly owing to the work of Leonard Hill, who, believing the variations to be entirely passive, summed up his views in the paradox that "the cerebral vasomotor nerves lie in the splanchnic area." Although the cerebral circulation differs from the circulation elsewhere in many ways, and particularly in its passive dependence upon external circulatory changes, it is now generally admitted to be also subject to active vasomotor control, although the final demonstration of this by direct physiological experiment is still lacking.

Harvey Cushing noted that epinephrin will blanch the pial vessels over the area of its application, and that faradic stimulation will do the same. Brodie and Ferrier found that on injecting adrenalin into the basilar artery of the removed brain the outflow from the torn sinuses was diminished or completely stopped according to the quantity injected.

During the course of an experiment which Professor Sherrington kindly gave me the opportunity of witnessing, I was struck by the fact that when a faradic stimulus of sufficient strength to evoke epileptiform movements was applied to the cortex, the pia-mater could be seen to blanch, and it naturally occurred to me to wonder whether epileptiform attacks starting from a traumatic lesion may not be accompanied or even initiated by a similar blanching of the cortex.

The evidence in favour of vasomotor activity in the brain is abundant, and it seems incredible that pathological variations in the cerebral circulation should not be associated with definite clinical manifestations. The characteristic rapidity and suddenness of vascular changes, and their characteristic tendency to recurrence, would allow of recurrent phases of disordered cerebral function, and on this hypothesis many of the phenomena of epilepsy can be fully explained.

The cerebral cortex has been raked over and over again by successive generations of neuro-histologists without yielding up the secret of the cause of idiopathic epilepsy, and it may be that the search, transferred from this barren field to that of the autonomic nervous system, will prove more fruitful.

Before discussing the part played by gross lesions in focal epilepsy, it is necessary to refer briefly to the effects of electrical stimulation of the cortex.

In man the effects of such stimulation appear to resemble closely those obtained from anthropoids, but as the recorded observations are comparatively few, and not primarily experimental, this need not be referred to further, except for one important point. Fedor Krause, in 1911, had the opportunity of stimulating the cortex of a patient who was the subject of Jacksonian epilepsy without gross visible lesion. He found that faradization of the epileptic zone elicited fits, whereas from the rest of the motor cortex only the corresponding movements were obtained, identical currents being used, and the duration of the stimuli being the same in each case.

In this connection an interesting and probably unique experience recorded by Sherrington is of considerable importance. Having by chance found amongst his laboratory animals a monkey which was the subject of epileptiform attacks, he took the opportunity of investigating the effect of cortical stimulation. The spontaneous attacks had begun in the left angle of the mouth (or perhaps in the tongue), and had spread to the rest of the face, neck, left arm, left leg, and then to the right limbs. Attacks could be induced by the taking of a large morsel

of food into the mouth. A minimal faradic stimulus applied to the tongue area of the right hemisphere provoked a tongue movement which almost immediately became clonic and epileptoid, meaning by the latter that it continued as a series of movements after the faradic stimulus of the cortex had been withdrawn. If the stimulus were continued for a few seconds the epileptoid movement spread from the tongue, and would occasionally involve all the facial muscles on the left side, but it never spread beyond the face. In the left hemisphere the tongue area yielded epileptic discharges easily, though not so readily as did the tongue area of the right cortex, nor did the movements spread. Sherrington remarks that it must be remembered that limited epileptiform discharge is, in the monkey, usually obtained by prolonged or quickly repeated faradic stimulations of almost any point in the motor cortex, and that therefore the result observed with the tongue area of this animal was of a quantitative rather than a qualitative nature. Further, that the exceptional readiness with which tongue epilepsy could be evoked was remarkable because, in his experience, the tongue area is not one from which epilepsy is usually easily elicited. The results of cortical stimulation in this epileptic monkey fall into line with those obtained by Krause in his epileptic human subject.

We require, however, an explanation of the fact that at least in the highest apes (and probably in man, to judge from Krause's observations) electrical stimulation is incapable of provoking a Jacksonian fit of any magnitude. Even in Sherrington's epileptic monkey: "By no persistence of the faradization could the epileptoid movement be made to extend beyond the face. It never, unlike the natural seizures observed in the animal, spread to the neck or limbs even of the same side, let alone the opposite." We know that in experimental work the depth of the narcosis affects the results in a marked degree, and it is possible that it is an important factor in determining the differences noted. It may be, too, that higher control, whether consciously exerted or not, accounts for differences in the extent to which the cortex responds to the stimulus of a focal lesion. It is a matter of common observation that patients can often arrest the spread of clonic spasms by holding firmly the affected limb at the commencement of a fit, by which manœuvre controlling influences are doubtless directed to the cortex involved, so as to neutralize the effect of the abnormal stimulation.

Horsley and Schäfer many years ago demonstrated the fact that the excitability of the cortex is increased by repeated stimulation, so that a weaker current will evoke a spread. This was confirmed and ampli-

fied by Leyton and Sherrington, who, in the course of their work upon the cerebral cortex, elicited many additional facts bearing upon focal epilepsy. Great variations were observed among individual animals in the ease with which a Jacksonian "march" could be provoked by the faradization of a cortical motor point. Not only is the threshold of excitability lowered by faradization, but the area from which a given movement can be obtained may be gradually extended. Thus after provoking movements of the lips, for example, by stimulating a cortical point just in front of the central sulcus, the same movement may be elicited by faradization of a previously inexcitable point more anteriorly situated. Further, faradization of the post-central cortex may facilitate the elicitation of movement from certain points in the precentral cortex at about the same horizontal level.

In this connection the following experience is of interest. Working with Dr. Gordon Holmes I had the opportunity of observing the effect of faradic stimulation of the cortex in a patient who for several years had been the subject of frequent focal fits resulting from a traumatic lesion. This lesion involved an area of the cortex, some 4 cm. in diameter, both behind and in front of the Rolandic sulcus, at about the level of the temporal crest. The stimulation was carried out in order if possible to identify the point of origin of the fits, and the technique employed was the same as that used by Sherrington. For obvious reasons the observations could not be made with the completeness of a physiological experiment, but there was no doubt as to the readiness with which epileptoid movements could be evoked from many points, some of which were far distant from the cortical area of normal representation.

Leyton and Sherrington have named the movements obtained by electrical excitation of the cortex "fractional" movements, and remark that they are in themselves co-ordinate and seem to form parts of complex purposive acts.

Electrical stimulation of the cortex, even when carried out with every possible refinement of technique, must be the grossest caricature of the natural stimuli which provoke purposive movements. Yet such electrical stimuli applied to the precentral cortex in man can be made to evoke definite movements, limited in extent and bearing a striking resemblance to simple natural movements. Although, as Ferrier first pointed out, these movements exhibit purposive co-ordination, they are not purposive movements, and in this respect resemble those of mild epileptiform attacks.

A Jacksonian fit is merely a series of fractional movements, devoid of purpose, and determined in point of sequence only by the relative anatomical position of the cortical areas in which they are represented. In other words, a Jacksonian fit is made up of a series of fractional acts, which, although in themselves co-ordinate, lack the mutual co-ordination of natural purposive acts. This mutual incoordination of fractional acts is illustrated by the biting of the tongue, due to the unnatural sequence of tongue protrusion and jaw closure. In this respect the movements of epilepsy resemble the effects provoked by electrical stimulation, which even when so applied as to evoke a "march" or sequence of movements, does not appear ever to call into action the peculiar synthetizing function of the cortex defined by Sherrington. No series of fractional movements so combined as to bring about a recognizable purposive act, even of a simple character, has, so far as I am able to ascertain, been recorded as resulting from electrical stimulation of any part of the cortex. In fact no more intelligent movements can be obtained from the cortex than from the mid-brain; they differ in character rather than in purpose, the one set taking the rhythmical form characteristic of movements obtained from the cortex, and the other lacking that particular nature. It seems to be not impossible therefore that the disturbance of cortical function, whether caused by experimental stimulation or by a gross pathological lesion, may, in part at least, be due to a lowering of the normal inhibition, and that the movements which result may be of the nature of release phenomena. On such a conception the epilepsy resulting from pathological lesions can be brought into line with that which can be induced experimentally.

The association between gunshot injuries of the brain and the development of fits is so obviously a case of cause and effect that I will refer to them before touching on the subject of fits associated with morbid lesions, and on those of idiopathic epilepsy.

The local morbid conditions underlying the fits which result from gunshot wounds of the head fall into three main groups.—

(1) Recent lesions such as local contusion associated with small hæmorrhages and œdema; or the more gross disruptive effects of a penetrating wound.

(2) Inflammatory lesions due to recrudescent sepsis, where a latent infection becomes active and gives rise to an area of softening, or to an actual abscess.

(3) Cicatrices binding the scalp to the brain and membranes through a cranial defect.

As regards the first two groups, the early fits, namely those which occur in the first few days after the injury, as well as those connected with recrudescent sepsis, are clearly associated with vascular disturbances due to direct damage to blood-vessels, thrombosis, inflammation and œdema; they continue only during the period of acute circulatory disorder, and cease when that period comes to an end.

The third group, with cicatrices binding the scalp to the damaged brain, is by far the most important, and it is to the cases included in this category that I wish to draw particular attention.

A year ago the Re-survey Boards of the Ministry of Pensions had, in the previous twelve months, examined more than 25,000 old cases of gunshot wound of the head. Excluding those examined more than once during that period, the number of individuals was approximately 18,000, amongst whom nearly 800 (or 4½ per cent.) were the subjects of epileptic fits. A very large number of such patients present a cranial defect through which the scalp adheres to the membranes and the damaged brain. Penetration of the dura at the time of the wound entailed not only gross direct damage to the underlying brain and its vessels, but also invasion of the injured tissues by micro-organisms. The consequent inflammation resulted in further destruction of nerve tissue as well as a greater disturbance of the vascular supply, both from thrombosis at the time and from strangulation of vessels later by fibrous tissue, the amount of which would to a large extent be proportional to the intensity and duration of the preceding inflammation. Further, the penetrating wound is inevitably followed by adhesions between the surface of the brain and the scalp.

It may reasonably be assumed that the rich blood supply of the normal cortex provides a wide margin of safety against accidental variations, whilst the relatively avascular scarred cortex possesses a smaller margin of nutritional safety. In these circumstances local circulatory disturbances would be likely to arise from accidental causes. One such cause which I believe to be of the very greatest importance, depends upon the fixation of the brain at the point of damage to the overlying membranes and scalp, and is occasioned by the cerebral movements.

There is no doubt that the normal brain obeys the law of gravity, and alters its position with varying positions of the head. Every organ of the body possesses a degree of mobility proportional to the extent to which it is covered by a serous membrane separating it from the wall of the cavity which contains it. The serous membranes of the thoracic

and abdominal cavities, as well as those which line the joints and the tendon sheaths, result from and facilitate movement. The brain is no exception; the serous cavity lying between the dura and the arachnoid can fulfil no other purpose; the intense pain occasioned by any movement of the head in meningitis is analogous to that of pleurisy. Whenever the skull and dura are widely opened during an operation, movements of the brain can be demonstrated by altering the position of the head, and although their extent is probably exaggerated by reason of the different physical conditions which obtain in the open as compared with the closed skull, there is no reason to suppose that all movement is absent when the skull is whole. Indeed J. Luys has demonstrated the fact of cerebral mobility by means of frozen sections, and Gavoy has, by means of a specially designed cerebral kinesiometer, been able to measure excursions of from three to nine millimetres.

When a brain, attached to the scalp by adhesions, attempts to move in response to a change of posture, it is prevented from doing so from a pull at the point of anchorage; this pull either mechanically or, possibly, by causing a reflex vasoconstriction, may well produce a momentary local anæmia in the damaged brain, and so initiate a fit.

Seeing, however, that only a very small number of patients with anchored brain develop epilepsy, it is obvious that the local lesion is but one of the links in the ætiological chain. Clearly a given stimulus is not effective for all brains, and it is necessary to assume in those patients who do develop fits a tendency to epilepsy resulting from what is vaguely called a relatively low degree of stability of the nervous tissue.

I have been fortunate enough to encounter a case which illustrates this important point remarkably well. A man, 35 years old, had at the age of 15 been accidentally wounded in the forehead by a revolver bullet. He was operated upon shortly after the accident, and was in hospital about fifteen weeks. He afterwards remained perfectly well in every way, joined the Motor Transport Service in 1915, and was sent to France where he served until 1919. He received no wound or other injury, but was often under shell fire and bombing. His first fit occurred after six months' service, and between that time and the date of his admission to hospital in 1920 he had about two dozen fits, all of which occurred during sleep, except two. The fits associated with frontal injuries of this character not infrequently occur during sleep, and they are, if the explanation which I have suggested is correct, to be attributed to the sudden changes of posture which may occur during sleep. This patient, in the two attacks which occurred in the daytime, fell unconscious,

without warning, and remained so for some minutes. He did not bite the tongue nor urinate; he suffered neither from headaches nor giddiness. There was a small irregular circular opening in the right frontal bone, into which the brain bulged on stooping forward. No abnormal neurological signs were detected.

The interpretation of this case seems to be that, although for twenty years this man had had a cranial defect through which the brain was anchored to the scalp, the local lesion had been insufficient to cause a fit until the nervous stability had been lowered by the stress of warfare. In May, 1920, an operation was performed. The scalp was dissected free from the dura, and the dura was separated from the bone for an inch or more beyond the edge of the opening. A thin sheet of celluloid was placed over the dura, extending some distance between it and the inner surface of the skull. A thicker sheet of celluloid was used to close the bony opening. Some ten weeks later he had a fit, whilst asleep, and since then no further fit has occurred.

In cases of this class the aim of an operation is twofold, first to ensure a permanent separation of the brain from the surface at the point of damage, so as to restore as far as possible its mobility; and secondly to close the bony opening so as to restore the physical conditions under which the cerebral circulation normally works. Both of these objects can be attained by the use of celluloid, the technique of which procedure will I hope form the subject of a future communication.

I and my assistants at the Tooting Pensions Hospital have now operated upon more than 200 cases by this method. About 120 of these patients suffered from fits. The time has not yet arrived for passing a judgment upon the results, but I may say that so far they are remarkably promising, even in some cases where the fits had occurred over a long period. Nor is it possible to estimate fairly the extent to which the beneficial results are due to the other treatment carried out at the same time, for in all cases the patients have been kept in hospital for from two to six months after the operation, and treated regularly with small doses of bromide.

It seems probable, to judge from our experience, that better results may be expected in cases where the fits have been infrequent, and have not occurred over a long period of time. Nevertheless, satisfactory results are not impossible even in long-standing cases, as shown by the following example:—

A man of 21 had, when 7 years of age, received a severe cranio-

cerebral injury in the left parietal region, which resulted in gross hemiparesis. A large area of bone had been removed, and the damaged brain had become adherent to the scalp. The brain pulled upon the scalp, which became concave when the patient lay upon his right side; the brain bulged when the head was inclined to the left.

Eleven years after the accident he began to suffer from fits, the origin of which was attributed to a blow upon the head in the region of the cranial defect. When admitted to St. Thomas's Hospital he had had fits for three years, for the first year about once a fortnight, for the second year about three times a week, and for the third year about once a month. On one occasion he had twenty-five fits in a single day. He was operated upon by the celluloid method and carefully treated for several months afterwards. Two years have now elapsed since the operation, during which time he has had only two fits. He has taken no drugs for a year, has improved greatly in general health, and is able to do some light work.

The claim of surgery to participate in the treatment of fits resulting from injuries to the skull and brain may fairly be regarded as well established, not only on the ground that a reasonable explanation exists of the mechanism by which they are evoked, but also because we now possess a good deal of evidence to show that the fits can in many cases be either abolished, or very considerably reduced in number and severity, by an operation which succeeds in removing or at least modifying, the local exciting cause. This evidence, now in course of preparation, I purpose bringing forward in the near future.

Leaving now the subject of epileptiform attacks due to gross injuries, we must approach the question as to what contribution surgery has to offer towards the treatment of fits other than those of traumatic origin, whether associated or not with local lesions of the brain. For the purpose of examining this question, I would divide these cases into three groups:—

The *first group* comprises all the cases in which there exists a lesion both lending itself to accurate localization and allowing of a reasonable surmise as to its nature. Many tumours, both innocent and malignant, as well as abscesses, cysts, tuberculomata and gummata, can be included in this category. Whilst any of these lesions may constitute the starting point of an epileptic attack, it must not be forgotton that there is no gross lesion, whether traumatic or otherwise, which necessarily causes fits. On the contrary, the proportion of patients with any such lesion who develop fits is small. As already mentioned, the incidence of

epilepsy amongst pensioners with old gunshot wounds of the brain does not at the present time reach 5 per cent.

The frequency, however, with which fits are associated with tumour of the brain is perhaps somewhat striking. I have recently looked through the notes of 270 cases of brain tumour, exclusive of cerebellar and pituitary cases, upon which I have operated. I find that no fewer than eighty-two patients, or 30 per cent., exhibited fits of a focal character. Grouping regionally the tumours associated with fits, I find that exactly half were situated precentrally, and of the remaining 41 22 were post-central, 12 temporal and 7 occipital. Of the whole 82 tumours, 65 were malignant and 17 were endotheliomata.

In no fewer than 40 per cent. the fit was the first symptom of which the patient complained. The fit was the first symptom in a rather larger proportion of the endotheliomata than of the gliomata, but the difference is not, in my opinion, sufficiently striking to be of any great value in diagnosis.

There can be no doubt, I think, that the nearer the tumour, or the surrounding area of cerebral softening, approaches the central sulcus, the more likely are fits to occur.

The exact nature of the stimulus by which a tumour or other gross lesion elicits evidence of cortical disturbance, whether of a motor or of a sensory character, is not easy to define. To say that a lesion "irritates" the brain merely creates fresh difficulties by raising the question of the exact meaning of irritation. Used in its usual sense irritation implies activity, but a lesion such as a bony boss resulting from a depressed fracture cannot be regarded as an active agent. If the brain from time to time accidentally, or continually on account of its respiratory and cardiac movements, impinges upon such a bony boss, changes conducive to epilepsy might occur. A tumour might be in a sense "active" from alterations in size or tension due to variations in its blood content, or to such sudden gross change as that caused by a hæmorrhage into it. Active inflammatory changes also occur from time to time in the membranes bounding a cortical tumour, whilst naturally vascular disturbances are a prominent feature where frankly inflammatory lesions are concerned. In the immediate neighbourhood of many of these lesions the appearances are such as to suggest that rapid or sudden alterations of vascularity could readily take place, and such changes seem to me to constitute the one common factor which is capable of explaining the associated epileptiform movements, and so bringing their causation into line with that of other forms of epilepsy, whether traumatic or apparently spontaneous.

In the surgical treatment of the cases included in this first category, namely of recognizable and localizable gross lesions, the operation is naturally directed primarily to dealing with the tumour or other lesion, and the result as regards the fits, as well as other symptoms, will depend upon its nature and the completeness with which it can be removed. When a simple tumour can be completely removed, or an abscess shelled out entire with its capsule, the outlook is good. When an abscess has to be drained, fits are not so likely to be abolished, since the drainage necessitates anchorage of the brain to the surface, which is in itself, as I have endeavoured to show, provocative of epilepsy.

The *second group* comprises those cases in which fits of a definitely focal character occur, but in which the nature of the lesion, if any, cannot be ascertained without actual inspection of the brain. Operation, which is fully justified in many of these cases, must necessarily be primarily of an exploratory character. In spite of the fact, however, that the technique of cranial operations has been so greatly improved, we must not fall into the error of putting a cerebral exploration quite on a level with an abdominal exploration, or undertake it with the same confidence that, even if nothing is found, no harm will result. By such exploratory operations I have found tumours, meningeal cysts, on one occasion a nævoid angeioma, and on another occasion a small abscess, all of which were capable of being satisfactorily dealt with.

On the other hand, a wide exposure of the cortex may reveal no visible lesion of any kind; the brain may appear to be absolutely normal both to inspection and palpation. In such a case the question naturally arises as to whether that portion of the cortex in which the initial movement is represented, having been defined by electrical excitation, may be excised. The effects of cortical excision upon function are, thanks to Horsley, Sherrington, Marinesco and others, well known; but the results of such excisions in patients suffering from focal fits without visible lesion have not, even in the hands of the most expert operators, proved very encouraging.

Cushing, from an experience of some fifteen cases, writes as follows: "Some had unexpectedly resulted in cures; one or two which promised to be especially favourable owing to the circumscribed nature of the initial movement (thumb and corner of mouth) were not benefited."

Krause gives details of three cases of focal epilepsy without visible lesion in which he excised what he calls the "primary spasming centre." One patient had had no fit some months later; one eight years later only had occasional twitchings; and one died shortly after the operation in status epilepticus.

Krause attributes the failure of cortical excisions in the hands of others to the localization having been made on anatomical grounds and not by electrical excitation. He believed the resultant scars to be of no importance, and not provocative of any spasm or symptoms whatever.

It is clear that in determining the advisability of performing cortical excision the resultant disabilities would be of less account in dealing with focal epilepsy starting in parts already deprived of their function. The published cases are too few and too insufficiently recorded to afford good ground for any definite conclusions. But the subject cannot be altogether dismissed without further investigation.

The *third group*, namely that of the so-called idiopathic epilepsies, includes all the cases in which the fits can neither be ascribed to any ascertainable organic lesion, nor possess any features pointing to a definite focal origin. I have searched in vain amongst the many records of operations for epilepsy, some purely fantastic, some based upon a more or less reasonable conception of its causation, for any encouragement to pursue further the quest for an operative procedure which holds out any prospect of benefiting the sufferer from idiopathic epilepsy. It may be unwise to say that the future will reveal nothing fresh in this direction, but it is at any rate certain that, at the present time, surgery has nothing to offer in the treatment of general idiopathic epilepsy.

In some other forms of epilepsy surgery may, as I have indicated, be made to play a useful part, but no operation can alone be expected to effect a cure. I am, however, convinced that, in properly selected cases, surgical treatment can be of great value in lessening the task demanded of medicinal and other therapeutic measures. If these measures cannot be carried out rigorously over a prolonged period, then, for the good of the patient and the credit of surgery, it were better to refrain from operating.

Cynical friends have told me that operations upon the nervous system do but illustrate the truth that hope ever triumphs over experience. The surgeon must be an optimist, but his optimism need not be that of the ostrich. It should be based upon the expectation that inquiry into the causation of those diseases which he seeks to remedy will eventually furnish the clue to their successful treatment. The search may lead him into deep waters, and this must be my excuse for venturing so far out of my depth as to address a gathering of neurologists on such a subject as epilepsy.

Notices of Recent Publications

Die vergleichende Anatomie des Nervensystems der Wirbeltiere und des Menschen. Von Dr. C. U. Ariëns Kappers. SS. 624. Mit 326 Figuren und 3 farbigen Tafeln. Haarlem: Bohn.

The establishment of the Central Institute for Brain Research in Amsterdam some years ago was a welcome experiment to all who are interested in the anatomy and development of the nervous system. And it became even more welcome when it was evident that, under the leadership of Dr. Ariëns Kappers, it would devote itself largely to the study of comparative nervous anatomy. Such an institute had the special opportunity to collect a body of workers who, in addition to producing individual essays on the phylogenetic development and comparative anatomy of separate portions and structures of the brain, would, under the direction of a capable chief, slowly collate and co-ordinate the knowledge that had been hitherto acquired so that we might ultimately obtain a comprehensive view, not only of the morphology of the brain, but also of the causes and factors that have determined its form in the higher vertebrates. The desirability of this is obvious since the anatomy and the adaptation to function of many structures in the body are comprehensible only when their evolutionary development is known, and this is particularly so in the nervous system.

There has been no lack of individual contributions to the comparative anatomy of separate portions of the nervous system, particularly by Continental and American workers, but the want has become acute of a textbook that would embody all the present knowledge in an accessible form for those who are unable to follow the contributions of each worker, or to sort out their results and integrate them critically into an intelligible whole. In this statement we are not unmindful of the well-known textbooks by Edinger, who must be justly regarded as father of this science, but the last edition of his book is several years old; and great as Edinger's achievements were, he was not, owing to his peculiar and original abilities, the most fitted person to harvest the enormous field he had sown. Smaller textbooks, as those by Herrick and Johnson, have been unquestionably useful as outlines or introductions, but their plan and compass precluded a full or adequate treatment of the subject.

That this want will be supplied by Kappers' book there can be no doubt. In the first section, which has already appeared, more than 600 pages are devoted to the histology and arrangement of nervous elements, and to the comparative anatomy of the spinal cord and the medulla oblongata. The present edition is appearing in German, but though English students may

regret that their language was not chosen, those with any knowledge of the German tongue will find no difficulty in reading Kappers' simple and lucid pages, or in following his well illustrated descriptions and his concise but always intelligible arguments.

This volume must not be, however, regarded merely as a textbook on comparative anatomy, for the human nervous system is dealt with as fully as in most books devoted to it alone. In fact its chief value to the neurologist is that the descriptions of the various structures found in the lower animals are so planned that they lead up to and simplify the study of the nervous system of man.

The first fifty pages, which contain a concise account of the histology of the nervous elements and supporting tissues, will probably afford less interest to the student than the remaining portions of this volume, though the author's critical review of many points still under discussion give it a distinct value, as well as make it a necessary prelude to the anatomical chapters.

In the next section the factors which determine the forms and connections of neurons are considered. Here we are introduced to the theory of neurobiotaxis on which Kappers has already written exclusively, but never so clearly and logically. The scope of a textbook and the opportunity it gives the author of reviewing the whole field of his experience, has enabled him to put his hypothesis in a simpler and more cogent form than when he attempted to illustrate it by some particular structure or system. And it must be admitted that, as presented in these pages, neurobiotaxis is more acceptable as a general law in the development of the nervous system than it appeared to be in some of the author's earlier papers. It is also pleasing to find that throughout the volume its application is not unduly forced, as frequently happens in the case of a newly discovered principle of such importance.

The author is not so happy in the distinction which he attempts to make between what he terms "vital" and "gnostic" functions, in the treatment of the lower levels of the nervous system at least. He uses "vital" as an alternative term to "protopathic," and "gnostic" as an equivalent of "epicritic," which in itself is an unfortunate choice, as these terms have been already employed with other meanings. Further, it seems very questionable whether such a distinction is possible in the lowest vertebrates, or in functions so primitive and elementary that they are subserved mainly by spinal and bulbar reflexes. The "vital functions" of the spinal afferent nerves are enumerated as the appreciation of pain, touch, and high and low temperatures, i.e., those perceptions to which Head has applied the term "protopathic," but a differentiation that describes gravi-statik as the "vital" (protopathic), and hearing as the "gnostic" (epicritic), function of the octavus nerve is liable to cause confusion.

The chapter on the comparative anatomy of the spinal cord affords an extraordinarily interesting review of the phylogenetic development of this segment of the nervous system from amphioxus to man. Here we can see not merely the increase in complexity of its structure as we ascend the animal

scale, but the manner in which new systems have developed within it, and its variations with the morphology and mode of life of the animal. The third chapter introduces us to the more difficult subject of the comparative anatomy of the medulla oblongata, though here too the systematic way in which the subject is treated, and the judicious use of diagrams and illustrations, make the descriptions as simple as they possibly could be. The constitution and connections of the branchial nerves of the fishes is regarded as one of the chief factors that determine the structure of the medulla in the vertebrate series, and the modifications that they or their derivates undergo in the course of the vertebrate evolution is made remarkably clear in the detailed description of their morphology in all the chief classes. This may be illustrated by the manner in which the anatomy of the afferent fibres of the facial, glossopharyngeal and vagal nerves is worked out, especially in their relation to the gustatory system. The cutaneous or somato-sensory components of these three nerves join the descending trigeminal root in the medulla, and end in the same mass of grey matter as it. In fishes their visceral and gustatory fibres enter the medulla and terminate together at the level of their entry in the viscero-sensory area. The earliest differentiation is found in the amphibians, where descending fibres of these roots first form a fasciculus solitarius; this contains viscero-sensory fibres only and has no connection with the gustatory system, as its size bears no relation to the development of the peripheral taste-organs, but corresponds to the sensory endowment of the upper alimentary and the respiratory tracts. As the non-specialized viscero-sensory fibres become segregated in the fasciculus solitarius the gustatory fibres of these three nerves pass to more dorsally and mesially placed nuclei in the floor of the fourth ventricle. The latter are identified with the nucleus intercalatus Staderini of mammals, which is regarded by Kappers as the chief lower gustatory centre. On the other hand, the grey matter, which is hypertrophied in the bony fishes into the lobus facialis and there receives the bulky afferent facial fibres supplying the taste-organs situated on the surface of the body, disappears in the higher vertebrates in which the cutaneous taste buds are no longer found.

The fourth chapter is devoted to the anatomy of the lateral-line and octavus system, and the fifth to the anatomy of the motor mechanisms of the brain-stem.

In the final chapter of this volume the reticular nuclei of the brain-stem and the inferior olives are studied in detail. In the lower fibres the reticular nuclei are discrete collections of large cells, generally related in position to the motor nuclei, but in the higher vertebrates many of them shift from this position, usually moving towards the afferent centres from which they receive impulses. The majority of their axons pass spinalwards, usually through the central longitudinal bundle. Their connections show that they are adapted to correlate impressions received from various sources and transmit them by their spinalward axons to the motor effector centres. They can be consequently regarded as co-ordinating mechanisms which collect or sort out stimuli with a common

or allied end-effect. In the higher vertebrates the reticular nuclei acquire a large proportion of smaller cells. In some instances, as in the nucleus ruber, the new cells have a cerebral connection, but most of the other reticular nuclei reserve their original function of motor co-ordinating centres of a lower level.

The inferior olives are equally ancient structures in phylogenetic history. Probably present in the most primitive fishes, they are certainly distinct structures from the selachians to man, though they undergo various modifications. The most important of these is the relatively greater growth of the main olive as compared with the accessory olives in the higher vertebrates, a change that is probably connected with the evolution of the mammalian cerebellum. All researches that have been undertaken on the olives indicate that their chief efferent connection is with the cerebellum, but we are still more or less ignorant of the sources of their afferent impulses. Kappers' suggestion that it may be mainly by collaterals from the afferent systems of the cord and medulla that they come, is interesting.

The reviewer cannot leave this volume without a word of praise for the manner in which it has been produced. The type is clear, the paper is excellent, misprints and typographical errors are rare, and the illustrations are most admirably reproduced. A very complete index, and a bibliography at the end of each chapter to the subjects treated therein, add to the value of the book.

GORDON HOLMES.

The Essentials of Mental Measurement. By Dr. WILLIAM BROWN, M.A., M.D., D.Sc. (Wilde Reader in Psychology in the University of Oxford), and Professor GODFREY H. THOMSON, D.Sc., Ph.D. (Professor of Education, Armstrong College, Newcastle-upon-Tyne). Cambridge University Press, 1921.

The publishers describe this new volume of *The Cambridge Psychological Library* as "a revised and expanded edition of Dr. Brown's original work, bearing the same title, published in 1911, together with a large amount of new material by Dr. Godfrey H. Thomson." Indeed, Dr. Brown's well known book has been more than doubled in size. Of the ten chapters in the work before us, five are entirely or almost entirely new, while two more have been expanded and altered, leaving only three substantially in the form in which they were first published a decade ago. The changes are wholly the work of Professor Thomson.

Dual in origin, the book is also dual in constitution. But it must not be supposed that the two distinguished authors are respectively responsible for the two chief ingredients. These are, firstly, an account of the principal mathematical methods that have been applied to statistics obtained by psychological and other experiments, and, secondly, a contribution to the discussion of general factors—and particularly of the "single general factor"—

in mental measurements. Especially in its former aspect, the book is of high value and fully deserves a place, not only in the "Cambridge Psychological Library," but also in the library of every University, College, or other institution where psychology is studied or applied. In its other aspect, the book is somewhat less satisfactory, although it has certainly advanced matters by calling for a reconsideration of one of the steps in the argument which Hart and Spearman employed (in 1912) for demonstrating the existence of a general factor in each of fourteen series of published tests, dating from various periods up to thirty years back.

The value of the book as an introduction to the mathematics of mental measurement has been enhanced by the addition of an excellent summary of the elementary theory of probability, including several interesting examples that are not to be found elsewhere. Another new chapter will be welcomed for the account it gives of Karl Pearson's fundamental work on frequency curves. The usefulness of the book might, however, have been further increased by including a little more information on some of the subjects treated. For example, the correlation between two variables is defined (pp. 101 and 109) in algebraic terms ($r = S(xy)/N\sigma_1\sigma_2$); and, although it is stated that "if the regression is linear, r is a measure of the degree of dependence between x and y," the simple geometrical aspect of correlation is never explained. Thus, whenever the relation between x and y is linear (as it always is in the case of two correlated variables, each of which is distributed according to the normal probability law), x and y may be regarded as the projections of *the same* variable line OP and two fixed lines, Ox and Oy respectively, while the correlation between x and y is, quite simply, the cosine of the angle between Ox and Oy. In other words, this correlation measures the average deviation in x (or y) corresponding to unit deviation in y (or x). Again, the "partial correlation" between x and y with z constant (where z is a third variable measuring the projectional OP on a third fixed line Oz) is just the cosine of the angle between the projection of Ox and Oy on a plane perpendicular to Oz. If geometrical interpretations of this kind had been taken into account there would have been no occasion for certain criticisms of details that we must make later. . . . Among other matters that might usefully have been included are Pearson's geometrical interpretations of his formulæ for correlations influenced by selection (p. 135); and a fuller account of the paper by Pearson and Filon (*Phil. Trans.*, 1898, cxci, A, pp. 229-311) of which important use is made on pp. 184 *et seq.*

But the other aspect of the book will be of more immediate interest to most readers. The authors' contribution to the theory of general ability may therefore be discussed, and perhaps criticized, at greater length than their admirable synopsis of the essential mathematics of mental measurement. It should not, however, be forgotten that the criticism that follows applies only to a comparatively small part of the book under review.

The fundamental mathematical proposition in the theory of general ability is to the effect that if the correlations between the measures of a number n

(larger than three) of qualities, each of which is distributed according to the normal probability law, form a perfect hierarchy, then the measure of each of these qualities is a vector compound of a single general factor, g (that is common to all the n qualities), and of a specific factor (that is peculiar to the particular quality in question), while g and the n specific factors are independent of each other. Mathematically expressed, the proposition is that if $q_1, q_2, \ldots q_n$ are the measures, in suitable units, of these n different qualities, and if the correlations (r_{st}, &c., where r_{st} is a correlation between q_s and q_t) satisfy the $\frac{1}{2}\ n\ (n-3)$ *independent conditions for a (perfect) hierarchy—namely that* $r_{as}/r_{at} = r_{bs}/r_{bt}$ . . . . . (1)

for all different values of $a, b, \ldots s, t, \ldots$ from 1 to n inclusive—then we can write—

$$q_s = g \cos \theta_s + \xi_s \sin \theta_s. \quad (s = 1, 2, \ldots n) \ldots (2)$$

or

$$q_s = r_{sg}\, g + \sqrt{1-r_{sg}^2}\ \xi_s. \quad (s = 1, 2, \ldots n) \ldots (3)$$

where $g, \xi_1, \xi_2, \ldots \xi_n$ are $n + 1$ independent factors, each of which is distributed according to the normal probability law, and where g is *the single general factor* common to all the q's, and ξ_s is a specific factor peculiar to q_s, and where r_{sg} is the correlation between q_s and g.

This definition of the single general factor is not given in the book under review; nor is the above proposition explicitly stated, although mention is made of the converse proposition—that if there is a single general factor then the conditions (1) will be satisfied. Moreover, these conditions, given on p. 165, are nowhere employed explicitly to define a (perfect) hierarchy. It can only be the absence of these definitions that renders possible such a statement as that the deduction of "The Theory of General Ability, or the Theory of Two Factors" [equation (3) above] "from the occurrence of 'hierarchical' order among the correlation coefficients is invalid" (p. 154). The statement to which the authors intended to give expression can hardly have been a denial of the certain fact that, where hierarchical order is perfect, there is a single general factor. They doubtless meant that when the hierarchical order is imperfect it is explicable otherwise than by means of a general factor that will be more or less dominant, together with group factors that will be more or less insignificant according to the degree of imperfection in the hierarchy. But their phrase, as it stands, is surely misleading.

To the same cause—lack of precise definition of the terms employed—we would attribute certain statements made in the course of the third section of Chapter VII (pp. 138 to 144). The section is concerned with three correlated variables. In such a case the number ($\frac{1}{2}\ n.\ n-3$) of independent conditions that have to be satisfied in order that the correlations may form a hierarchy reduces to zero, since $n = 3$. Consequently, it is *always* possible to find a 'single general factor' common to three correlated variables. And yet (on p. 140) the authors say "there is no general factor whatever," and, again, "we cannot say with certainty whether a general factor exists or

not." General factors—that are not the single general factor just defined above—can, in fact, be found for three correlated variables in a trebly infinite number of ways; for, given any three correlated variables, q_1, q_2, q_3 (distributed according to the normal law with the same probable error), it is possible to choose, in a trebly infinite number of ways, three independent variables (also distributed according to the normal law with still the same probable error) ξ, η, ζ, such that—

$$\left.\begin{aligned} q_1 &= \lambda_1 \xi + \mu_1 \eta + \nu_1 \zeta \\ q_2 &= \lambda_2 \xi + \mu_2 \eta + \nu_2 \zeta \\ q_3 &= \lambda_3 \xi + \mu_3 \eta + \nu_3 \zeta \end{aligned}\right\} \quad \ldots\ldots\ldots (4)$$

and it is always possible to choose one of them, say ξ, so that λ_1, λ_2 and λ_3 are all finite; or, in other words, so that ξ is a general factor of q_1, q_2 and q_3. If in the example in which the book before us says, "there is no general factor whatever," we put q_1, q_2, q_3 proportional to the scores obtained by throwing n purple dice (whose score, reduced to standard deviation, is p), n green dice (reduced score, g) and n orange dice (reduced score, o), so that q_1 is the reduced score of purple and orange, q_2 of orange and green, and q_3 of green and purple, we have—

$$\left.\begin{aligned} q_1 &= \frac{1}{\sqrt{2}}\, p + \frac{1}{\sqrt{2}}\, o \\ q_2 &= \frac{1}{\sqrt{2}}\, g + \frac{1}{\sqrt{2}}\, o \\ q_3 &= \frac{1}{\sqrt{2}}\, g + \frac{1}{\sqrt{2}}\, p \end{aligned}\right\} \quad \ldots\ldots\ldots (5)$$

in which, indeed, no general factor is apparent. But it is there, none the less. For it is, of course, possible here (as in every other case) to choose independent variables, ξ, η, ζ, such that any one of them, say ξ, is a general factor. For example, we may choose q_1 itself (putting $\xi = q_1$ in equation (4)) as one of the independent variables, and the plane $\xi O \eta$ to contain Oq_2. Then equation (5) becomes—

$$\left.\begin{aligned} q_1 &= \xi \\ q_2 &= \tfrac{1}{2}\, \xi + \frac{\sqrt{3}}{2}\, \eta \\ q_3 &= \tfrac{1}{2}\, \xi + \frac{1}{2\sqrt{3}}\, \eta + \sqrt{\frac{2}{3}}\, \zeta \end{aligned}\right\} \quad \ldots (6)$$

revealing $\xi = \frac{1}{\sqrt{2}}\,(p + o)$ as a general factor of q_1, q_2 and q_3; $\eta = \frac{1}{\sqrt{6}}\,(2g - p + o)$ as a group factor of q_2 and q_3; and $\zeta = -\frac{1}{\sqrt{3}}(g + p - o)$ as a specific factor of q_3. And this is only one of an infinite number of ways in which q_1, q_2 and q_3 may be expressed, by means of equations (4), as linear functions of three independent factors of which one is a general factor. And yet the authors say that "we cannot say with certainty whether a general factor exists or not." They add (p. 142) that the condition that three correlated variables, representing dice scores, may have a general factor is that—

$$r_{23}^2 + r_{31}^2 + r_{12}^2 + 2\, r_{23}\, r_{31}\, r_{12}$$

shall be greater than unity. But this is really the condition that it will be possible to choose ξ in equation (4), not only so that λ_1, λ_2 and λ_3 shall all be finite, making ξ a general factor (which can always be done) but *also so that certain very special conditions*, namely—

$$\frac{\mu_2}{\mu_1} = \frac{\mu_1}{\lambda_1} \text{ or } o\,; \quad \frac{\mu_3}{\lambda_3} = \frac{\mu_1}{\lambda_1} \text{ or } o\,; \quad \frac{\nu_2}{\lambda_2} = \frac{\nu_1}{\lambda_1} \text{ or } o\,; \text{ and } \frac{\nu_3}{\lambda_3} = \frac{\nu_1}{\lambda_1} \text{ or } o \quad \ldots (7)$$

shall be satisfied at the same time.

Nothing is said about these very special conditions, except that "it may not be out of place to remind ourselves again that this, though true of the arrangements of dice we are considering, may not be true in the same sense of other phenomena, e.g., biological or mental phenomena" (p. 142). Yet it is these special conditions, whose natures are not examined, that cause the error here and cause further misapprehensions in Chapter X (pp. 175 *et seq.*). Their origin is to be found in the fact that, while the general expression for any one (say q_s) of n correlated variables (q_1, q_2 . . . q_n), each of which is distributed according to the normal law with the same probable error, in terms of N (where N is not less than n) independent variables (x_1, x_2, . . ., x_N), each of which is distributed according to the normal law with still the same probable error, is—

$$q_s = {}_sl_1\, x_1 + {}_sl_2\, x_2 + \ldots + {}_sl_N x_N \; (s = 1, 2, \ldots n) \ldots (8)$$

where—

$$1 = {}_sl_1^2 + {}_sl_2^2 + \ldots + {}_sl_N^2 \qquad (s = 1, 2, \ldots n) \ldots (9)$$

the expression for q_s when x_1 . . . x_N represent dice scores is—

$$q_s = \frac{1}{\sqrt{Nc_s}} \times (\text{sum of } Nc_s \text{ whole } x\text{'s}) \; (s = 1, 2, \ldots n) \ldots (10)$$

where c_s is the proportion of the whole number of x's that enter into qs. In other words, every l in equation (8) becomes in the special case of dice throws either zero or the same as every other l that is finite.

Now suppose that the x's in the several q's are chosen by picking out Nc_1 x's at random to form q_1; then putting these back and picking out Nc_2 x's to form q_2; and so on. Then it has been shown that this may be done so that the proportion of x's common to any group of q's and to no others (*i.e.*, occurring in each member of the group and in no other q's), would be equal to the probability that any particular x was common to that group and to no others; and that, in this case, the correlation, r_{st}, between q_s and q_t is $r_{st} = \sqrt{c_s c_t}$ so that $r_{as}/r_{at} = r_{bs}/r_{bt}$ for all values of a, b, . . . s, t, . . . from 1 to n. In this case, therefore, there is a perfect hierarchy. It follows that the q's are compounded, according to equation (3) above, of a single general factor and specific factors only. It may be shown that the same tends to be true when the x's in qs are selected as in the example described on pp. 175 and 176 of the book under review. The resulting approximation to hierarchical order is due to the very special form of equation (10) as compared with the general equation (8) and to the manner in which the x's in equation (10) are selected. And yet the

authors conclude that since "Dr. Hart and Professor Spearman would . . . claim the hierarchy as being a sample of a very perfect one," which it is, "this example goes far towards shaking confidence in their criterion." We cannot agree. This example does not affect the consequences that follow when Hart and Spearman's conditions are satisfied—that is when the correlation of every pair of columns in a square table of correlation coefficients is ± 1; still less does it throw any doubt on the truth of the fundamental proposition that when there is a perfect hierarchy there is a single general factor and specific factors only.

But the formula employed by Hart and Spearman in order to correct "observed" coefficients of correlation may be faulty, as our authors argue. In that case further experimental tests of dissimilar mental qualities in large numbers of individuals will still be needed in order to settle the question whether the correlations of these qualities form hierarchies (as was very approximately the case with both the series of tests which Cyril Burt made on school boys in Oxford) and so demonstrate that there is present a single general factor in every quality tested.

We emphasize the need for testing dissimilar qualities in these experiments, because group factors will always be present in every group of similar qualities. In intellectual qualities, for example, it is convenient to recognize a group factor, "cleverness," and in moral qualities another group factor having some close relation to "purpose." These group factors are, by definition, independent of each other and of the single general factor. They may be important. But the book under review surely errs when it says (p. 173) that "Mr. Maxwell Garnett . . . concludes that in addition to the single general factor of Professor Spearman, there are two large group factors which are practically general." It is true that Webb, who first noticed one of them, called it a "second general factor." But would he or anyone else expect either of these group factors to appear in qualities—in sensory discrimination, for example—which are outside their own special groups, but which may still depend upon the single general factor?

It should be noticed that Thomson's sampling theory of ability (equation (10)), and Spearman's theory of general ability (equation (3)), are by no means mutually exclusive. If further experimental investigations confirm the existing evidence that the correlations of sufficiently dissimilar mental tests tend to form hierarchies, we shall be able to say with equal truth that the several qualities may be regarded as compounded (according to equation (3)) of a single general factor—general ability—and specific factors only; or that each quality is composed (according to the all-or-none law in equation (10)) of a sample of a very large number of distinct elements. Time will show which of these two conceptions will prove to be the more fruitful.

MAXWELL GARNETT.

Syphilis und Nervensystem. Von Dr. MAX NONNE. Vierte, neu-durchgesehene und vermehrte Auflage, mit 169 Abbildungen im Text. Pp. 1,019. Berlin: Karger, 1921.

This well-known work, consisting originally of a series of lectures, has been thoroughly revised and brought up to date. It still remains the most comprehensive account of the effects of syphilitic infection of the nervous system, and we have not been able to discover any aspect of the subject that is not thoroughly considered in the light of recent research and clinical experience. It is well printed on remarkably good paper and should be in the possession of every neurologist.

Pathology of the Nervous System. By E. FARQUHAR BUZZARD, M.D., and J. GODWIN GREENFIELD, M.D. Pp. 334, with numerous illustrations. London: Constable, 1921.

Both the senior student and practitioner have long desired a short but authoritative account of those pathological changes which accompany common diseases of the nervous system. This book exactly fills such a want. It is clearly written, and the information given is accurate and in advance of that found in textbooks. The illustrations are admirable; they are excellently reproduced from original photographs of specimens prepared by the authors. Each chapter is followed by a short series of references to such literature bearing on the subject as is accessible in any medical library. This little book can be highly recommended to all who are anxious to acquire a sound knowledge of the pathological anatomy of the nervous system.

The Basis of Psychiatry (Psychobiological Medicine). By ALBERT C. BUCKLEY, M.D. Pp. 447, with 79 illustrations. Philadelphia and London: Lippincott, 1920.

The first part is devoted to a somewhat commonplace account of the functions of the central nervous system and the biological phenomena underlying heredity. This is followed by a classification of mental diseases according to their causes, manic-depressive insanity and paranoia excepted; for instance, under alcoholic psychoses nine separate varieties are described. In the chapter dealing with methods of examination we are not told how to make observations on the mental state of the patient, but there is a full account of the technique of the Wassermann reaction. Dementia præcox and paranoia, those touchstones of a textbook on psychiatry, are described purely from the aspect of the patient's external behaviour and no attempt is made to analyse the various psychical factors which go to make up his mental state.

The Clinical Examination of the Nervous System. By G. H. Monrad-Krohn, M.D.Christiania, M.R.C.P.London, Lecturer in Neurology at the Royal Frederick University, Christiania. With a foreword by T. Grainger Stewart, M.D., F.R.C.P., Physician to Out-Patients, National Hospital for the Paralysed and Epileptic. Pp. 135, with 12 diagrams. London: H. K. Lewis, 1921.

This little volume contains a useful description of the more important methods to be employed in the investigation of mental and nervous functions. The author's choice of tests is excellent and he has incorporated in this book the results of much recent neurological work. He rightly lays stress upon the necessity for linking up psychiatry with neurology, and insists on the importance of investigating the mental functions during the routine examination of the nervous system. The student will find this volume a most useful companion both during his work in the wards and subsequently in practice.

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To those who are not members of the Neurological Section of the Royal Society of Medicine the price is 8s. 6d. net, and the volume may be obtained through any bookseller.

EDITOR.